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OF THE

(Ophthalmological Division)

OF THE

American Academy of
Ophthalmology and
Oto=Laryngology,

AT ITS

Eighth Annual Session,

HELD AT

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Indianapolis, Ind., April 10, 11 and 12, 1903.



AMERICAN JOURNAL OF OPHTHALMOLOGY,
St. Louis, Mo.

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The Tenth Meeting will be held in Denver, Col., August 24, 25, 26, 1904.

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MINUTES OF THE EIGHTH ANNUAL MEETING
—OF THE—
**American Academy of Ophthalmology and
Oto-Laryngology,**

—HELD AT—
INDIANAPOLIS, APRIL 9th, 10th and 11th, 1903.

THE meeting was called to order at 9 a. m. by the President, D. W. L. Ballenger.

Dr. J. J. Kyle, Chairman of the Arrangement Committee, greeted the members of the Association, and after some further remarks stated that on Thursday evening a smoker with vaudeville performance at the Columbia Club, and on Friday evening a banquet at the new Dennison Hotel had been arranged for.

The President then extended his greeting to the assembly, and referring to the fact that he had been born in Indiana, stated that he was proud to be a Hoosier.

Next the regular order of business was taken up as announced in the programme.

The reading of the minutes of last meeting was dispensed with. The Secretary and the Treasurer's reports were presented and accepted. A number of telegrams were read and answered.

After this the President introduced Mr. Bookwalter, Mayor of the city of Indianapolis, who in a very witty and enjoyable manner welcomed the Association most heartily.

Dr. Casey A. Wood, as Chairman of the Committee on a New Constitution and By-Laws, reported what had been done by this committee, and this report was adopted in almost the exact form as presented. It included the change of the name of Western Ophthalmic and Oto-Laryngologic Association to the "American Academy of Ophthalmology and Oto-Laryngology."

Forty-eight new members were elected, the names having been recommended by the Committee on Election.

The Nominating Committee reported the following names for officers of the Association :

President, . . . Dr. Edward Jackson, Denver, Col.
 1st Vice-President, Dr. D. S. Reynolds, Louisville, Ky.
 2nd Vice-President, Dr. J. J. Kyle, Indianapolis, Ind.
 3rd Vice-President, Dr. J. W. Murphy, Cincinnati, O.
 Secretary, . . . Dr. D. T. Vail, Cincinnati, O.
 Treasurer, . . . Dr. O. J. Stein, Chicago, Ill.

Members of the Council :

Dr. Edward Jackson, Denver, Col.
 Dr. Adolf Alt, St. Louis, Mo.
 Dr. W. L. Ballenger, Chicago, Ill.
 Dr. C. A. Wood, Chicago, Ill.
 Dr. C. A. Holmes, Cincinnati, O.

On motion of Drs. W. L. Ballenger and D. T. Vail the following resolution was adopted :

Resolved, That it is the sense of the American Academy of Ophthalmology and Oto-Laryngology that street railway companies should be encouraged by public sentiment to introduce noiseless wheels and other appliances to overcome the horrible noise which is so characteristic of many of the systems now in operation in our large cities.

Resolved, That in our opinion thousands in our hospitals and homes, who are ill, need this courtesy, as they are rendered more ill by the din and clatter of the cars passing along the streets.

At the motion of Dr. M. A. Goldstein a contract for the publication of this association's transactions was entered into with Drs. Alt and Goldstein.

Dr. Alt gave notice that at the next annual meeting he would move that a member having announced a paper on the program and failing to read it personally, except for very valid reasons, such paper shall not be printed in the transactions.

Dr. W. L. Ballenger moved the following resolution, which was adopted :

That the American Academy of Ophthalmology and Oto-

Laryngology deems it wise to correspond with all other American associations devoted to the consideration of diseases of the eye, ear, nose and throat, with a view of establishing a federation of the said societies. The said federation to meet in joint session once every five years for the consideration of scientific and other subjects pertaining to the eye, ear, nose and throat.

After the usual votes of thanks had been offered and responded to, the Academy adjourned to meet on August 24, 25 and 26, 1904, at Denver, Col.

THE PRESIDENT'S ADDRESS.*

WILLIAM LINCOLN BALLENGER, M.D.

CHICAGO.

THE development of the Mississippi Valley and the Great West was well advanced when this society was organized eight years ago. Many prosperous and growing cities of 10,000 and upwards were standing as milestones along the highways of commerce, agriculture and social progress. Schools, colleges, universities, libraries, hospitals, churches, cathedrals and great mills and shops of industry were everywhere to be found. Railroads threaded the whole country as the veins and arteries of a living body.

The symphony of a new civilization filled the world with its music. The great countries of Europe heard the strains and many thousands of her people migrated hither annually. They quickly caught the spirit of the new song and became integral parts of the great hosts filling the valleys, plains and mountains of this territory.

We may say, then, speaking of the middle and great West as a whole, that only within the last decade has it had a symmetrical and uniform development. Prior to this time great urban centers were few and so widely separated that there was little opportunity for the social and scientific intercourse so essential to the well-being of any body of men.

The European clinics and those of New York and Philadelphia supplied the post-graduate training which prepared the physician for a practice limited to diseases of the eye, ear, nose and throat. The number thus limiting their practice was, up to this time, comparatively small. As the commercial and social conditions became more and more crystalized the demand for trained oculists, aurists and laryngologists became so great that hundreds of them entered the new field. In many places the territory was as yet but imperfectly organized into county and State medical societies, and as for local societies devoted to diseases of the eye, ear, nose

* Read at the banquet tendered the Academy by the profession of Indianapolis.

and throat, they were unknown. The means of meeting other oculists and aurists upon a ground of common interest were not afforded, except in the sectional meetings of the American Medical Association. These were not adequate, as the places of meeting were usually so remote from the great majority of those interested that many found it inconvenient or impossible to attend. They were, therefore, left to carve out their destiny alone.

Isolation leads to individualism. This is well enough so long as the propensities of the individual lead in the right direction. If, however, they lead him to loose and questionable methods of conducting his professional work we must then conclude that isolation and individualism are bad.

The law of self-preservation makes it desirable that men engaged in similar lines of work should conduct themselves in an honorable manner, as for one to do otherwise would reflect discredit upon, and create distrust of the whole fraternity. It seems, therefore, that isolation is detrimental to the best interests of the individual physician, his confrères and to the community he serves.

In the great cities this objection is met to a certain extent by the large number of men engaged in special practice. But even there the problem is not wholly solved, as only local interests are considered and subserved. The scattered or widely separated oculists and aurists are not taken into consideration. It would appear, therefore, that the best interests of ophthalmologists, otologists and laryngologists were not adequately provided for prior to the organization of this society. Nor were they fully met then, as its field of work was still sectional and incomplete.

It is true that national associations of ophthalmology, otology and laryngology were in existence then, but their requirements for membership were such as to practically exclude all but those of wide reputation. As a large percentage of the specialists in the middle and far west were located in small cities with no clinical, hospital or college facilities, their opportunities for distinguishing themselves were limited. They were thus shut off from membership in these great organizations. Hence we return to the proposition, that, there was inadequate provision for the promotion of the best

interests of a large number of oculists and aurists in this portion of the country.

The need for such a society was recognized by the founders of the sections on ophthalmology, otology and laryngology in the American Medical Association. They have done much toward a solution of the economical, sociological and ethical problems peculiar to the wide distribution of specialists throughout this broad land of ours.

Where there are three or more oculists and aurists in a city it would be to their advantage to band themselves together for social and scientific purposes. Petty jealousies arising from imperfect knowledge of each other would largely disappear, leaving in their place a mutual esteem and confidence that would sweeten their lives and clear the cobwebs of distrust from their brains. From jealous rivals they would become friendly ones, each profiting in the financial as well as in the social amenities of life.

It was no doubt in pursuance of this need that the Western Ophthalmological, Otological, Rhinological and Laryngological Association was organized. Its founders may have builded better than they knew, as the present status of the society has already demonstrated the supreme wisdom of the procedure.

The Association has passed through the period of infancy and childhood and has just reached its majority or state of legal adulthood. At first its name was enrolled in full in the old family records as the Western Ophthalmological, Otological, Rhinological and Laryngological Association. Not a euphonious name, it is true, but one sufficiently distinctive to differentate it from the other societies of the same species. A little later, when it had arrived at the state of lusty childhood, its name was suitably shortened to the Western Ophthalmologic and Oto-Laryngologic Association, to meet the exigencies of its boyhood period. Having now reached its majority it was yesterday christened the "American Academy of Ophthalmology and Oto-Laryngology," a title suggestive of its present dignity and importance.

The new title is indicative of its present place in the field of ophthalmologic, otologic and laryngologic endeavor. When an infant in arms its scope was quite circumscribed,

and as a child it was somewhat enlarged; now that it has reached adulthood its workshop is the entire so-called American world. In thus enlarging its borders it is not encroaching upon, nor is it in any sense a rival of, any other organization in existence. Each has its peculiar mission to accomplish. The American Ophthalmological, the American Laryngological, the American Otological and the American Laryngological, Rhinological and Otological Associations each properly limits its membership to men already somewhat distinguished, thus closing its door to a large number who are, nevertheless, in every way worthy of recognition as reputable practitioners of ophthalmology and otology. Our society meets this want, and in this lies the chief reason for its existence.

The personnel of our membership is such as would do honor to any society in the world. It would be invidious for me to single out any particular names, and, indeed, the roll would be too long. An analysis of the names enrolled in our roster would, however, reveal the fact that a majority are already distinguished men, while a large number of the remainder are men of marked ability only awaiting the time and opportunity to add luster and honor to their chosen profession.

The new constitution recognizes the desirability of encouraging the untried worker and provides for him in the article on membership. He enters upon his work with more or less training (too often less than more), having nebulous ideas of his proper relationship to his confrères, the profession at large, and to the community he seeks to serve. It is just at this time that he is most in need of intercourse with his seniors in practice. From them he will, all unconsciously, learn the standards of attainments and of ethical conduct, which will most surely bring him honor and preferment in the profession. He will thus be enabled to avoid the dangers of individualism which so often wreck the careers of men of even great ability. Whether we will or not, we are, as individuals, but integral parts of a co-ordinate body, and as such we should adjust ourselves to the recognized standards and methods of work and expression. To go contrary to these standards and methods would bring disrepute upon the offender, as well as discredit upon the medical fraternity as a

whole. Hence in the new constitution the doors are opened to the novice in ophthalmology, otology, rhinology and laryngology on condition that he is a member of the American Medical Association and is of good repute in his local medical society. In the course of the next few years after his admission he will have ample opportunity to demonstrate his ability and moral worth to the profession, and may receive the approval and honorable recognition of this society by being elected to Fellowship in the same. The Membership period is probational and in a sense educational.

In seeking the recognition of his peers he presents a Fellowship thesis. If his individualism has been held subservient to the best interests and highest honor of the profession, and the thesis is of high order, his election to Fellowship is assured. In this way his scientific, practical and ethical faculties are enlarged and developed. It is not always to the best interests of the novice that he be at once officially recognized as the equal of his seniors in practice. Experience and endeavor should count for something, and the object of this constitutional provision is to encourage him to develop accurate powers of observation and to establish a true ethical relation to his associates in practice.

This is much better than to leave him out of the society until he has established a reputation. The temptations to professional irregularity are many, and if the precept and example of his peers and legitimate associates are withheld from him he may fall. The Academy opens its doors to him and believes it is not doing him a favor but a simple justice. On the other hand he may be helpful and inspiring to his seniors.

Many men come into our midst fresh from the fields of study and at once impress us with their superior mentality and training; but they have not yet proven their ability to adapt their individualism to the interests of the profession as an organic body. They may have ability equal to their peers and only need the ripening influence of experience and social contact to merit their full approval. In encouraging and guiding the younger men we are adding to the reputation of every other physician. Whatever discredits one belittles all, and what is to the credit of one is helpful to all.

We believe, therefore, that in making provision for the early entry of the young oculists and aurists into the Academy the interests of all are served.

Having attained Fellowship with us he feels the consciousness of having *earned* the esteem and confidence of his fellow-workers. Added dignity and increased self-respect give him a mental and moral poise which is calculated to make him all the more conservative.

A new era is dawning in the history of the national associations of ophthalmology and oto-laryngology. The time is fast coming when it will be desirable that there be a *federation* of all the associations now in existence. Each will maintain its separate organization, but they will unite at stated intervals for scientific, legislative and sociologic purposes. It is not desirable that any national association, devoted to the consideration of the diseases affecting the eye, ear, nose and throat, should cease to exist; nor so far as I know is it desirable that they should materially change their plan of organization. The spirit of the age is one of organization, the individual giving way to the composite, but this does not necessarily mean that the many associations now in existence should disband and unite in one organization, but that they should form a *federation* of all their interests and meet in joint session, say once every five years; and thus by a community of interests and purposes promote fellowship, American in its scope; combine their influence for the enactment of such legislation as may be deemed to the interest of the profession and to the people, and to further the progress of such sociologic problems as are constantly confronting the medical profession.

In conclusion I wish to say that I have but words of admiration and commendation for the other national associations devoted to ophthalmology, otology, rhinology and laryngology. I have the honor of membership in some of them and count it my chiefest distinction. Each fills a place peculiar to its form of organization. Our Academy has its mission, a need, not comprehended in the other societies.

All hail! then, to the Academy of Ophthalmology and Oto-Laryngology. Her past has been resplendent with brilliant achievements and exalted social amenities. All hail! to

the inspiration that led to its organization. All hail! to its efficient officers and members, who have so faithfully brought it to its present honorable position. All hail! and all hail! to the new constitution, where the novitiate and the physician of ripe experience may associate and interchange ideas and ideals.

Long may the American Academy of Ophthalmology and Oto-Laryngology live; and living, inspire the young to great achievements, and noble! The young men will grow older and wise, while the old and wise will halt by the way full of years and honor. Let us drink, then, to the health of the young, who will grow old and wise; then, let us fill to the brim and drink to the old and wise who are full of years and honor.

All hail! all hail! and all hail! to the Academy as she is to-day, and to her as she will be on the morrow.

ESSAYS AND DISCUSSIONS.

CLINICAL EXPERIENCES IN THE TREATMENT OF PHORIAS AND TROPIAS.

By J. ELLIOTT COLBURN, M.D.

FIRST I desire to call your attention to a case of heterophoria in a neurasthenic patient with the following history:

H. M. Male, aged 35 years, decorator by occupation. Mental confusion, pain in the orbital region following or attending the use of the eyes, inability to fix the eyes for any length of time upon his work or a printed page, vertigo and scintillating scotomata. Nutrition of the body good.

Refraction:—Vision equaled $\frac{20}{60}$ in the left, $\frac{20}{200}$ in the right. Under mydriasis, left equaled $\frac{20}{70}$, right $\frac{20}{200}$. With —1. spherical combined with a —1.25 cyl. ax. 180 for the left and —1.50 spherical with a —1.25 cyl. ax. 180 for the right, vision equaled $\frac{20}{20}$ in each eye.

Name, Mr. H. M.
No.

Date.....	Jan. 1903					Feb.		March						
	21	22	23	25	27	4	5	6	13	14	18	19	25	
Hyperph. R.....	1	0	0			0	0	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{3}{4}$	$1\frac{1}{4}$	0	3	0
Hyperph. L.....	0	0					0							
Esophor.....	9	9	5	0	0									
Esoph. in A.....	0													
Exophor.....	0							$2\frac{1}{4}$	$2\frac{1}{4}$	3	$2\frac{1}{2}$	4		0
Exoph. in A.....	3	8	7	8	8	8	$7\frac{1}{2}$	16				16		
Abduction.....		2	5				7		7			8		
Adduction.....			30				28		28			28		
Sursum, R.....	$1\frac{1}{2}$											4		
Sursum, L.....	$1\frac{1}{2}$											2		
Hyperph.....														
Esophoria.....	7													
Exophoria.....														
Rest Prisms.....	10 0 5					7	7		R.H. $\frac{3}{4}$		R.H. $1\frac{1}{2}$			
									Ex. 1		Ex. 1			

Remarks:..... (+2)

In this case the only satisfactory reading of the muscle condition was obtained after he had been for two months under the influence of moderate doses of bromides, arseniates and extracts of malt, with out of door life and light active exercise. The patient was found to have exophoria equaling 4° , in accommodation 15° , right hyperphoria $2\frac{1}{2}^{\circ}$ and a cyclophoria equaling -4° in the left eye.

I have long found it of advantage in the study of uncertain cases to use bromide of sodium in ten grain doses three times a day and have usually found that if true hyperphoria existed the results of the test would become uniform.

At the suggestion of Dr. Oscar King this patient was given arsenauo and fattening diet. The cyclo-exophoria was corrected by a tenotomy of the left externus when the hyperphoria gradually disappeared.

Three cases of general chorea, mother and her two daughters. There was no personal history in these cases which would indicate the cause of the condition and it was only by a study of the mother's history that I felt warranted in correcting the heterophoria in the children, hoping for a favorable result.

FIRST CASE. The mother, Mrs. C., aged 32 years, general health good from childhood with the exception of general chorea which began at her 12th year and continued with varying intensity until her 17th year, when she began to suffer from migraine and daily headaches. This condition obtained until she came to me in 1893. She was refracted and at that time was relieved to a slight degree of her headaches. The attacks of migraine and occasional frontal headaches were sufficiently troublesome to have her return to me a year later for review. I then found that the glasses as first given were correct but that she showed, as she had done at the time of her first visit, a left hyperphoria of 4° with occasional diplopia when fatigued. Ten days later the error equaled six degrees, and under rest prisms used for three hours the diplopia became constant. There was no declination and no lateral errors. The left hyperphoria was corrected by a graduated tenotomy, the first result of which was a right hyperphoria of one degree, which gradually disappeared.

Three months following the operation she reported com-

plete freedom from headache. I prescribed during the past year presbyopic correction.

Since the first operation she has used her eyes as she had never been able to do before, and without discomfort. Her general health, mental and physical, has improved.

Her two daughters began with chorea at twelve and ten years of age, though not at the same time, that is, they occurred about two years apart. Their errors of refraction were corrected, and later left hyperphoria 3° and $3\frac{1}{2}^{\circ}$ being corrected the chorea disappeared. Up to date of writing there has been no manifestation of headache or migraine.

A troublesome feature in these cases was the frequent occurrence during the tests and at other times of transient scotomata, not scintillating, lasting from just a moment to five or ten minutes. These attacks were never, so far as I could discover, preceded by a flash of light or followed by headache, usually occurred in one eye at a time, and were incomplete and irregular in form. After the heterophoria was corrected the attacks of scotoma gradually disappeared. It is likely that the condition resembled ophthalmic migraine and later, as in the mother's case, would have become pronounced.

The value of the rest prism and the development of the latent or total error is a question which interests everyone who enters upon the study of heterophoria. The error may be manifest or latent, either in part or totally. The construction of the head, the expression, pose and history may point to an error of direction, but every test result be negative. From the fact that the results of the tests are negative I do not judge that the patient is free from muscle imbalance, but that I have not been able to discover it by the usual tests, and may never be. As we can not put the muscle at rest and have to resort to other means of determining the nature of the trouble. Rest prisms may be used to aid in the detection of the error and the determination of the degree.

We now place prisms in position to partially correct manifest or to favor the supposed error. In an hour the test is again made and the manifest condition noted. Should the prism be rejected after repeated trials I should render a Scotch verdict of not proven. If, however, the prism was

not only tolerated but a slight increase, one or two degrees, accepted, a greater and greater amount may be brought out by slowly adding $\frac{1}{2}$, 1, 2 or more degrees as the tests indicate, always keeping a little below the error shown at the last reading. The following case will illustrate:

Miss H. D., aged 20 years, a stenographer, had been repeatedly refracted, under atropin gave a progressive myopia —1.50 each eye. Attempts were made to relieve a troublesome asthenopia, pain in the back of the head and beginning anæmia from malnutrition. The accompanying history chart can well illustrate the method used in the study of this case. In this case the readings were taken every hour or half hour:

Name, Miss H. D. Age 20. Stenographer.
Refraction, —1.75 corrected right and left.

Date.....	August 8						9	10	11	12			18	3
	A.M.					P.M.				A.M.	M	P.M.		
	9	10	10:30	11	11:30	1				9	10	11	12	1
Hyperph. R.	0	0	0	0	0	0								
Hyperph. L.														
Esophor	1	3	6	9	12	16	16	16	12	16	16	16	16	16
Esoph in A.														
Exodhor														
Exoph. in A.														
Abduction														
Adduction	3	3			3	3	3	2	2	2				
Sursum, R.	42	42			42		42	48	50	50				
Sursum, L.														
Hyperph.														
Esophoria		15			16	16				15				
Exophoria														
Rest Prisms.	1	2	5	9	11	15	0	8	0	0	15	16	16	17

Parallax
Test.

Red glass
Diplopia

P. Tenot. L-ft R-c. Int.

Remarks: No declination. Head posed to left.

My reason for securing the total result by a single tenotomy will be found in the pose of the head and the limitation of the abduction of the left eye. Five months subsequent to the operation the young woman reported that she was free from headache and able to do her work without discomfort. The phoria was completely relieved, and rest prisms used as at the time of the visit failed to develop an error greater than at the time of the last preceding visit.

The condition of heterotropia and heterophoria or voluntary or alternating tropia or phoria I have found difficult to study and still more difficult to treat. The nearer together the refractive conditions the less difficulty in correcting the error.

Amblyopia from whatever cause always complicates the study and final judgment as to the advisability of attempting a correction after the twentieth year of age. Eccentric pose of the head, while a suggestive aid to diagnosis, is a troublesome complication, as it prevents the best results in both tenotomies and advancements.

January, 1891, Miss E., aged 20 years, was referred to me by my friend, Dr. E. I. Kerlin, for persistent headache through and back of the eyes, back of the head and in the temples. The temple and occipital pains were almost constant. From the constant discomfort her general nutrition had been lowered and she was reduced in flesh and in blood count. As no other cause for the head pain was found by the family physician, an examination of her refraction was suggested. This examination was conducted under atropin mydriasis and right hyperopia. + 50 sph., left + .75 sph. + .13 cy. ax. 90° were found to correct her errors of refraction. Left hyperphoria equaled 2°, esophoria equaled 6°. Adduction equaled 14°, abduction equaled 10°. Right sursumduction equaled 1°, left sursumduction equaled 3°. No cyclophoria (Savage test). Correction for the error of refraction was given and modified at various times during the following three months, change of surroundings, a trip to the country, tonics, etc., etc., were ordered without in any way relieving the symptoms, though she could not be comfortable without her glasses. Seven months later the hyperphoria was corrected without benefit. Later the externi were tenotomized, resulting in a slight over-correction of the error equaling $\frac{1}{2}^{\circ}$. Glasses were again modified without relief. I now advised the patient that I was at my limit, so far as the eyes were concerned, and asked for a consultation first from an expert diagnostician and later by an ophthalmologist, if no general cause was found. During a general physical examination it developed that both kidneys were afloat and that after manipulation the patient was free from pain and discomfort, only to relapse as she moved about. This was a failure not of diagnosis and treatment of ocular errors, but of diagnosis of the conditions causing the nervous symptoms. Up to my last knowledge of the patient nothing had been done toward fixing the kidneys, and the patient is still a sufferer from eye

strain symptoms. One other case of floating kidney has come under my observation with symptoms simulating eye strain.

Complete Tenotomy for the Correction of a Phoria. Mr. M., aged 28 years, was referred by a neighboring practitioner with the following note:

The patient is coming to Chicago to make it his permanent home.

"I am referring a Mr. M. to you for the correction of a left hyperphoria, which was originally a left cataphoria, but owing to the slipping of my sutures the present condition resulted. The patient had 18 degrees of cataphoria at first and desiring to test the method proposed by an eastern ophthalmologist of cutting the tendon completely off in cases of four degrees or more, and thinking I was safe with that large amount, I followed the suggestion, with about six degrees remaining, but this gradually passed over to twenty-four degrees of the present trouble. I then attempted to advance the inferior rectus and got a good result, but the tissue did not seem to unite readily and my sutures were slipping, so I weakened the superior. The correction was only temporary when the hyperphoria gradually returned, and as a last resort I again advanced the inferior, but my sutures again slipped and the union was slow."

When the patient came to me, as nearly as I could make out, he had on the perimeter 24 (geometrical) of L hyperphoria. The thickening resulting from the last operation had not fully disappeared, but it was not until I had opened the conjunctiva that I realized that it would have been better to have delayed the operation until a later date. My attempt to secure the muscle in its proper position proved a partial failure, as in January just past I found 20° (prism) of error remaining, but by posing his head he was able to secure single vision. In January last I was able to reattach the muscle, leaving but one degree of left hyperphoria.

I can not from a personal experience condemn complete tenotomy for the correction of a phoria. I can only say that I do not feel justified in attempting it nor would I attempt a tenotomy of the inferior, either partial or complete. I have found it difficult to gauge the effect of an operation on the inferior muscle. Stevens has pointed out the difficulties at-

tending operations on these muscles. In my opinion from what I know of this class of cases an advancement is the better operation. While more difficult, the results are positive and, if properly executed, satisfactory.

Mr. L. K., aged 24 years, a student, had been refracted under atropin. Glasses had been prescribed and frequently changed. The asthenopia headache and general neurasthenia were in no way benefited, though he could not work without his correcting glasses.

Right +1.

Left +1. with +.75 cyl. ax. 90°.

A diagnosis of hyper-esophoria equaling 4° \perp 8° had been made and prisms given for its correction. With all this there was no lessening of his symptoms, and a year of out of door life was advised. As this would delay him in obtaining his degree he came to me in consultation. I found refraction and muscle imbalance as reported and advised a tenotomy for the correction of the hyperphoria and later the esophoria. The case was further considered and ten days later found a hyperphoria of 8° with a varying esophoria. Without the correction of the error of refraction I could not make out a declination either by Stevens or Savage tests.

A graduated tenotomy of the right superior rectus was done and an immediate measurement taken. There was but slight change in the phoria. Another investigation was made and it was found that a central band far back had escaped the Stevens hook. When this was severed an over-correction was obtained which necessitated the use of a restraining suture to prevent too great a displacement, as I desired to advance the muscle in the fellow eye. In this case the muscle was heavy in its center and the lateral wings were thin and unusually broad. The check band which prevented the recession of the muscle was thin and unusually broad in its ocular attachment.

The vertical plane of the right eye was nearly the same as that of the plane of the face, five degrees, while the corresponding plane of the left eye was thirty degrees, the normal in a symmetrical face being fifteen degrees. Plane B, see the *Journal of the American Med. Assn.*, Oct. 18, 1902. (Fig. 1).

In operating, particularly for the lateral errors, I have found it best to pay full respect to the total error as shown by the rest prisms, correcting it by advancement or tenotomy.

For the correction of cyclophoria I prefer in low degrees associated with hyperphoria the graduated tenotomy, while in high degrees of declination the advancement after Stevens' method, or as preferable to anchor the muscle to the tendon of the adjacent wing of the rectus muscle.

Mr. G., aged 32 years, had been examined and error of refraction corrected under atropia used four or five days. There was great difficulty experienced in correctly locating the axis of a low cylinder for the right eye, it being alternately received at 90, 75 or 105 degrees. There was a slight exo-

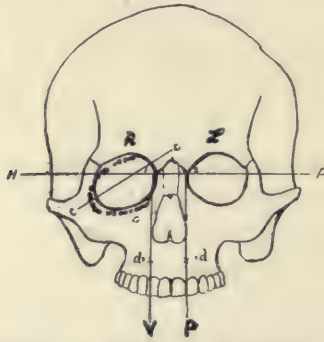


FIG. 1.—H P, Horizontal plane; 1, 2, attachments of the orbicularis palpebrarum; V P, verticale plane; d, d, canine fossa; c, c, direction of largest diameter of the orbit.

phoria at twenty feet and pronounced exophoria at the proximal point. A minus cyclophoria of five degrees was constantly observed under the Savage test. There was distortion of the page with correction before the eyes and alternate acceptance and comfort, and rejection and discomfort with the lenses at 90 degrees.

After some weeks observation, ocular gymnastics and general ocular rest the patient was operated after the Stevens method and the glasses accepted at 90 degrees without return of asthenopia or headache. In this case there had been in the later hours of the day a facial tic which has completely disappeared.

My first experiences with the Stevens method were not markedly successful, as I did not appreciate the importance

of placing the stitch in the stronger portion of the annular ligament.

It seems to me important to call attention to the fact that heterophoria in any form can not be studied or treated without the exercise of extreme patience, whether they prove to be operative or non-operative cases.

Dr. H. S. B. I called attention to this patient in June, 1902, at the meeting of the American Medical Association, and herewith offer the report then made and the supplementary report:

CASE 4.—Mr. B. Aged 24, robust general health, sturdy physique, has from early childhood suffered from headache,

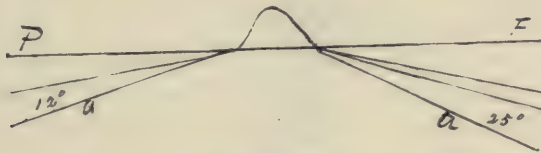


FIG. 2.—A. P. Left orbital angle; A. F. right.

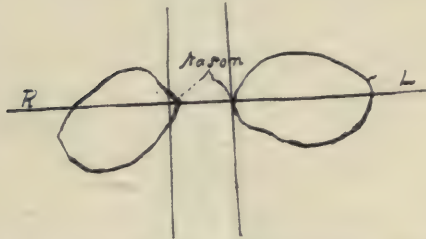


FIG. 3.—Non-symmetrical position of bases of orbits.

periodic asthenopia usually during school terms and has attacks of petit mal, but during the past three years these have been infrequent as he has been engaged in active out-of-door life. His military training has forced him to hold the head in nearly the primary position. He is always conscious of an effort to secure and maintain binocular single vision. With the slightest effort diplopia results and the disengaged eye either turns upward and outward or downward and outward, as the case may be, to a sufficient degree to rid the patient of the confusion of double vision. I have never been able to satisfactorily estimate the total error. Refraction R, V= $\frac{20}{20}$, L, V= $\frac{20}{20}$. Atropia R, +1.= $\frac{20}{20}$, L, +.75= $\frac{20}{20}$. Head in primary position, field of fixation normal, adduction =26,

abduction = 16. R sd. = ? L sd. = ? Orbital planes: Right eye plane A 25, plane B 20, declination = 25. Left eye plane A 12, plane B 10, C 15. You will observe that the plane A (Figs. 2 and 3) in the right eye has a greater angle by 13 than the left, and the plane B is greater by 10 (Figs. 4 and 5). We would class his head as broad and flat if viewed from the right side; if viewed from the left side, long and thin. When attention is relaxed the habitual pose of his head is to the left with the chin depressed. * An effort was made about six years ago

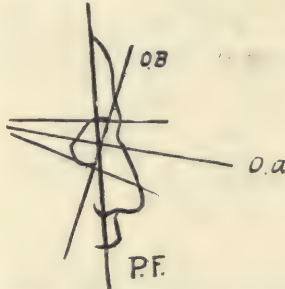


FIG. 4.—Right side of face; P. F. vertical plane of face; O. A. orbital axis; O. B. orbital base.

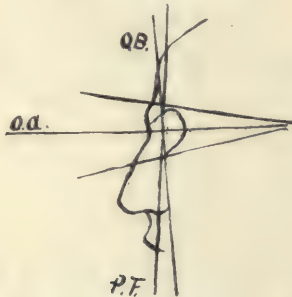


FIG. 5.—Left side of face; P. F. vertical plane of face; O. A. orbital axis; O. B. orbital base.

to correct the hyperphoria by a graduated tenotomy and advancement, but shortly after the tenotomies were made the patient was called into active service and the error has not been taken under consideration until quite recently.

The status of the case when he came to me again in the autumn of 1902 was as follows: Head in primary position. Perfect parallelism. Red glass showed left hyperphoria of nine degrees which quickly passed twenty degrees. Exophoria equalled 14. Could fuse by effort and fix with the left eye and the right would assume the hypophoric position equaling

9°. The Maddox rod, Stephens phorometer or parallax test gave about nine degrees of left hyperphoria. A partial tenotomy of the superior rectus reduced the error to 7°, and two months later an advancement of the right superior rectus gave perfect correction of the hyperphoria. Three months later a partial tenotomy of the right externus and an advancement of the right internus corrected the exophoria, giving him two degrees of esophoria.

In this case tenotomies in themselves were of little use. The advancements only were of avail. It must take months of careful use of the eyes to do away with the bad habits of fixation and the disturbance of the nerve centers governing the recti muscles. Training the muscles, by prism or other exercises, does not count for much. They have the power to overcome high prisms, but the effort resulting from sustained fixation under difficulties resulting from asymmetrical formation and direction of the orbits is too great even for well-trained nerve centers and muscles.

The basis for the error in this case was undoubtedly anatomical. And while an unusually sturdy physique and nervous make-up enabled him to do a large amount of work, yet with each added year he realized the increasing labor and discomfort of the handicap.

DISCUSSION.

DR. FERD C. HOTZ, Chicago.—The diagnosis and management of phoria cases is certainly one of the most perplexing problems oculists have to deal with. As the descriptions of the essayist of his careful and persistent examinations show, we cannot come to a conclusion in such cases by one or two examinations. There are so many points to be considered in relation to the muscles of the eye, as to whether they are a disturbing element; whether the discovered phoria is a real thing and one that we can change. That alone requires great care and patience and perseverance to find out; and before we have satisfied ourselves that it is actual, we certainly have no right to interfere, especially by the mechanical means of an operation. With our present facilities we can certainly discover phoria in almost every patient, just as we can discover a refractive error in almost every eye. The question is: Is the phoria a source of

trouble, an etiological factor in the complaints of the patient? If a doctor limits his observations to the eye and works out phoria only, without taking into consideration the numerous other conditions of the nervous system and of the whole body in every way, he is apt to encounter dangerous pitfalls and make most serious blunders. I wish to relate a few such observations which are most instructive to me. To the younger members it should be a warning not to look at the eye with blinders on.

Some time ago a college student consulted me with the history that he had been suffering with asthenopia, for the relief of which he had been submitted to three or four graduated tenotomies, but without relief. There was some esophoria present, two degrees. Both eyes were myopic. The fundus showed marked irritation of the choroid. I put him on a rest cure under atropine, and in four or five weeks he was able to use his eyes with ease.

Another case shortly afterwards in my clinic was that of a patient with slight paresis of the externus of the left eye. He said that three months before a tenotomy of the superior rectus had been performed on that eye—for the correction of what I do not know. The man had apparently perfect health, was strong, robust and intelligent. I made the remark to my class that a paresis of an ocular muscle coming on in a man of middle age in apparently good health, where we have no traumatism and no specific history, always ought to induce us to investigate as to the possibility of beginning locomotor-ataxy. When I made this man stand up with his eyes closed, he began to sway in a few seconds, so that he would have fallen had not the students supported him. The knee-jerk was entirely gone. The diagnosis of locomotor-ataxy was confirmed. In that case, operative interference with whatever might have been the matter with the superior or inferior rectus was absolutely uncalled for. But some doctors are sometimes so absorbed in what they see in the eye that they forget that the eye is, after all, not disconnected from the rest of the human body.

DR. W. L. DAYTON, Lincoln, Neb.—With all honor to my friend Dr. Hotz, who, I think, is an anti-phorist, I will say that I certainly believe in phorias. I feel just so sure as we can have talipes, that we can have an insufficiency of the ocular muscles. Patients certainly come to us with asymmetrical faces, and in

these cases we frequently find shortening of one muscle, or one set of muscles, or insufficiency of several sets. The question is, what shall we do, not, whether this condition exists. The question is whether the thing to do is a tenotomy or treatment of the systemic conditions. The question is as to the remedy to provide. I have served a rather long apprenticeship in graduated tenotomies, and I have advanced, resected and done several other methods in order to correct the condition I have found existing. I can certainly say that there is no operation I have found that will answer in all cases. We must select our cases for graduated tenotomies, resections and advancements.

DR. HOTZ.—It is a great surprise to me that my remarks were interpreted as my being opposed to operative treatment of heterophoria. This was not the purpose of my remarks. I cannot cover the entire ground in the few minutes allowed, but I simply took up one point in voicing a few words of caution. I fully acknowledge the existence of heterophoria. I am treating such cases and am not opposed to operation where needed.

DR. COLBURN (closing discussion).—I do not know that I have anything to offer in reply, as there has been, so far, no criticism of my statements. I do not feel like criticising the operation of complete tenotomy for heterophoria. I have never practiced it myself and do not know the results, but I would criticise the tenotomy on the inferior rectus muscle. You cannot estimate the effect of either advancement or tenotomy, and I would warn any one from touching in any way the inferior rectus muscle.

THREE ESSENTIAL POINTS IN THE OPERATION FOR CICATRICAL ECTROPIUM.

By F. C. HOTZ, M.D.,

Prof. of Ophthalmology and Otology in Rush Medical College, Chicago.

SOMEbody once has said blepharoplastic operations look very pretty—on paper, but in reality the results are anything but pleasing and satisfactory. The operations for cicatricial ectropium are not excepted from this criticism. The restored lids often look hideous, and still oftener are everted again after a few weeks or months. But we can overcome these drawbacks, if we pay strict attention to the following points:

1. The proper division and fixation of the skin flaps.
2. The selection of the most suitable material for covering the lids.

3. The shortening of the overstretched lid border.

1. *The Proper Division and Fixation of the Skin Flaps.* The greatest difficulty we have to contend with in the operations for cicatricial ectropium, is the tendency of the shrinking skin flaps to evert the lids again. As all transplanted flaps will undergo more or less shrinkage and as the lid border is least capable of offering any resistance to the traction of the shrinking flap, a re-eversion of some degree is almost inevitable as long as the transplanted flap is attached to the non-resisting lid border on the one side and to the non-yielding skin of the forehead or cheek on the other side, because the lid border is then exposed to the full traction force of the shrinking flap. It is evidently much easier for this flap to pull the lower lid down than to draw the skin of the cheek up, and therefore re-eversion is an especially common occurrence after the operation for ectropium of the lower lid.

It is plain, then, that to prevent the re-occurrence of ectropium we must place the lid border beyond the reach of this traction force. And this can be accomplished if instead of covering the whole wound with one skin flap we make use of two flaps, a small one which is to cover the lid surface only and which we will call the *lid flap*, and a larger one which is to be spread over the remaining wound area; and furthermore if we make provision that the contraction of the larger flap can have no effect upon the lid flap.

In ectropium of the upper lid this point is gained if the upper edge of the lid flap (Fig. 1 ba.) is firmly attached to the upper border (a) of the tarsus, while its lower edge (b) is united with the free margin. The lid flap is thus anchored above and below to the tarsus and its contraction cannot turn the lid over because to do so the traction force must have a fixed point of purchase outside of the lid. The shrinkage of the other skin flap (ac) which has its point of purchase (c) outside of the lid, however, cannot disturb the position of the lid margin, because its pulling force is expended entirely on the upper tarsal border (a); for, thanks to its firm union with this border, the lid flap cannot be stretched or drawn upward

by the contraction of the other flap and as long as the lid flap cannot be drawn up, the lid border is effectually guarded against re-eversion.

In ectropium of the lower lid, the same principle is adopted for the protection of the lid against the vicious traction of the shrinking flap. But we must bear in mind that the tarsus of the lower lid is very small and narrow and that normally the lid skin reaches farther down than the lower border of the tarsus, to a slight furrow a little above the infraorbital margin, (Arlt's tarso-malar furrow) where the integument

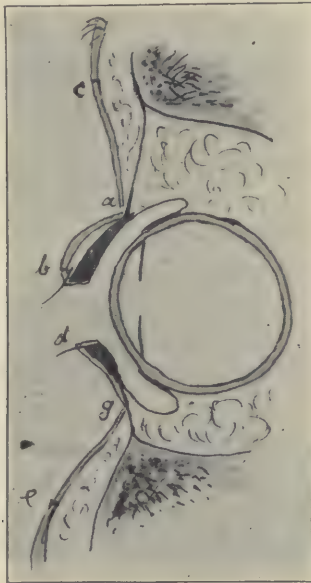


FIG. 1.

passes from the upright plane of the lid into the sloping surface of the cheek. This normal boundary line between the lower lid and cheek must be re-established by our division and fixation of the skin flaps; and the lid flap (dg) therefore must not be united with the lower border of the tarsus, but fastened to the tarso-orbital fascia (at g) in a line a little above the infraorbital margin. As the fascia is, as it were, the anatomical continuation of the tarsus, the lid flap adherent to the surface of the tarsus and fastened to the fascia cannot evert the lid. And the shrinkage of the large flap (ge) covering the wound of the cheek, is prevented from

pulling on the lid margin and causing re-eversion of the lid, because its traction cannot reach beyond the firm union of the lid flap and fascia. So strong is the resistance of this union that you may draw the skin of the cheek down as much as you please and you will not succeed in everting the lid.

2. *The Selection of the Most Suitable Material for Skin Flaps.* If it is our ambition to obtain the best possible results in ectropium operations—and nothing less ought to satisfy us—we cannot be contented with the mere reposition of the lids, but should strive also to restore as much as possible their natural appearance. The lids contribute more to the expression of the eye than the eyeball itself; and the slightest alteration of shape and outlines, or the least impediment of motion seriously mars the expression of the face. This is a very important point and should always be taken into account when we choose the material for our flaps. The normal integument of the lids is a thin and delicate skin of such perfect flexibility and adaptability that it allows perfect freedom of motion and accommodates itself readily to the numerous slight changes in contour so characteristic for the various facial expressions. In order, therefore, to gain a perfect cosmetic result we must select as a substitute for the lost lid skin, a material which possesses the same qualities; it must be thin, light and adaptable. It goes without saying that the thick skin of the forehead, temples or cheek does not possess these qualities; and therefore flaps from these regions should not be transplanted upon the lids. Besides the transplantation inflicts additional wounds upon the face and I hold we should avoid marring the face by unnecessary scars. The use of Wolfe's flaps taken from the arm avoids this latter objection, but they, too, are usually so thick that like the pedunculated flaps, they make a heavy, clumsy-looking lid. These two kinds of flaps are as well suited for a substitute of lid skin as shoe leather would be for kid gloves.

A good and suitable material for lid flaps which answers all the requirements is the cicatricial skin usually found in the immediate vicinity of extensive ectropium. I have shown in 1896* that this skin can be successfully transplanted and

*Journal of the American Medical Association. September 19, 1896, and Archives of Ophthalm. Vol. 25, No. 3.

makes a perfect lid skin. In ectropium of the lower lid this cicatricial skin can nearly always be utilized for the lid flap. In ectropium of the upper lid it can sometimes be used when the eyebrows are absent. If on account of the eyebrows the cicatricial skin is not available, a Thiersch graft is the only suitable material. It accommodates itself to the surface of the lid, brings out its contours perfectly and does not in the least interfere with the free movements of the lid.

3. *The Shortening of the Elongated Border of the Lower Lid.* As the diameter is shorter than the half circle, so in the case of complete ectropium the free lid margin of the lower lid turned from its almost straight line between the canthi into a long downward curve is elongated a good deal; and after being held in this abnormal state for a long while it will not recover its normal length when the lid is replaced. The elongated margin of the lower lid will not closely fit the curvature of the eyeball and will drop away from it, as in a senile ectropium. The reposition of the everted lower lid, therefore, cannot be perfect and permanent, unless the over-stretched lid margin is reduced to its proper length. In several instances I did not do that, because the lid margin appeared to lie in perfect apposition to the globe; but every time I had cause to regret this omission and had to correct my mistake by a second operation. I, therefore, regard the shortening of the elongated border of the lower lid a very essential point in the operation for cicatricial ectropium.

And now, after having so strongly emphasized the importance of these several points, I wish to indicate briefly the plan of operation based upon the views expressed in the foregoing remarks. As the operation is not alike in details for both lids, I believe it is better for a clear understanding to describe the method for each lid separately.

Technique of the Operation Upon the Upper Lid. If the eyebrows are partly absent and there is a good expanse of cicatricial skin above the everted lid, we cut from this cicatricial skin the lid flap in the following manner: **“From a point (a Fig. 2) about 5 millimetres above the inner canthus an incision is made obliquely upwards into the cicatricial skin and then continued in a curve downwards to a point (c) about*

*Archives of Ophthalm. Vol. 25.

5 millimetres from the external canthus. This incision outlines a large flap (abc) which is carefully dissected up from the underlying scar tissue as far as the lid border. The lid is then released by dissection from all cicatricial connections



FIG. 2.

until it can be easily turned down into its normal position; and now the edge (Fig. 3 ac) of the lid flap is fastened by silk sutures to the upper border of the tarsus."



FIG. 3.

If, however, on account of the eyebrows the lid flap cannot be taken from the cicatricial skin, we make an incision along the lid border and after the reposition of the lid cut from the arm a Thiersch graft of suitable size which is transported on the razor directly to the lid, spread out over its surface and fastened

by fine silk sutures to the upper tarsal border as well as to the wound edge of the free border. These sutures must be inserted with great care to make sure the edges of the flap do not roll in; their application therefore is very tedious work; but the time is well spent because the graft thus fixed is positively secured against being shifted from its place by any movement of the lid or by any accidental displacement of the dressing. After the lid flap is fastened (as in Fig. 3) the lid is drawn down as far as possible and held in this position by two ligatures passed through the free border and fixed on the cheek by plaster strips. This is done for the purpose of immobilizing the lid during the healing process and also for enlarging the wound (a, b, c), above

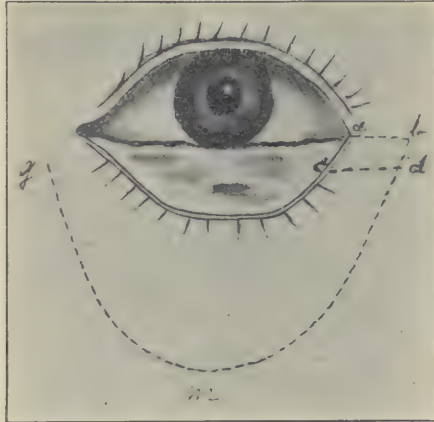


FIG. 4.

the lid to its fullest capacity. Over this wound a Thiersch graft is spread out so that its edges lap over the surrounding skin; no sutures are used.

TECHNIQUE OF THE OPERATION ON THE LOWER LID. Here the lid flap can always be procured from the cicatricial skin; but as this skin shrinks considerably as soon as it is dissected up we must take good care to cut the flap of very liberal dimensions. We begin the incision one centimetre below the inner canthus (Fig. 4g), carry it obliquely down into the cheek to a point (m) 2 to 3 centimetres below the centre of the everted lid margin; then we continue it in an oblique direction upwards and outwards to a point (b) even with and one centimetre from the outer canthus. This large flap (gmb) is then dissected up from

the underlying scar tissue and all cicatricial strands and bands are cut until the lid is freed and can be turned up. The next step is to reduce the overstretched lid margin to its proper length by removing a suitable piece of the lid (except the conjunctiva) near the outer canthus by the following incisions: from the canthus (a) transversely to (b), from (a) to (c) along the lid margin, and from (c) to (d) through the flap; the edges (cd) and (ab) are then united by two silk sutures. Now the lid is drawn up as far as possible and held in this position by two silk ligatures passed through the free margin and fastened on the forehead by adhesive plaster or collodion. This

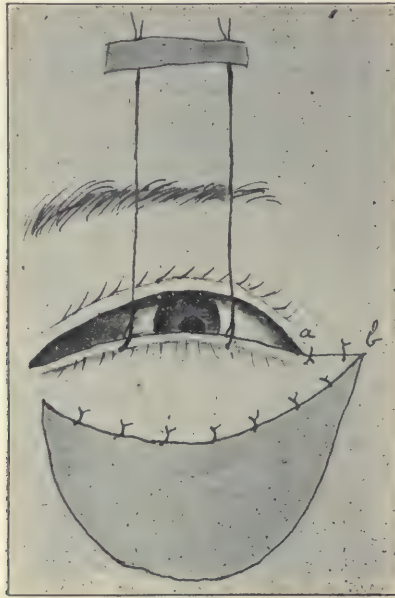


FIG. 5.

done the edge of the lid flap is anchored to the tarso-orbital fascia by silk sutures (Fig. 5). The lid flap should be evenly spread out, so as to be in perfect contact with the wound surface; but we must carefully avoid any undue stretching; and should we find that the flap is a trifle short and would be stretched if the sutures are placed very near the infraorbital margin, it is better to put them through the fascia 1 or 2 millimetres higher up. Finally the wound surface (gmb) below is covered with a Thiersch graft the edges of which are made to lap over the surrounding skin. No sutures.

THE AFTER TREATMENT is the same for the upper and lower lid. Strips of gutta percha protective are laid over the flaps, and upon these strips a gauze compress wrung out of warm boric acid solution; this is covered by a large, square piece of protective to prevent evaporation and over this is put a layer of cotton to maintain uniform warmth. This whole dressing is secured in place by a roller bandage and best left undisturbed for three days. Then it is carefully removed; the flaps are thoroughly cleansed, the overlapping edges of the large flap are trimmed off and a new dressing is put on which is changed every day or two as circumstances may require. At the end of the first week the ligatures and the sutures of the lid may be removed; during the second week some simple ointment (like borated vaseline) is applied daily on the flaps and after this period no further treatment is required.

DISCUSSION.

DR. CASEY WOOD.—I feel a debt of gratitude to Dr. Hotz for his suggestions in the surgery of the lid. The main thing in his experience of grafts is the selection of the skin. Dr. Hotz suggests that grafts be taken from the arm. If a Thiersch and not a Wolfe graft be employed, I endorse that statement. If the directions to use a Wolfe graft are followed, it must be remembered that the hair of our bodies is removed or its growth reduced to a minimum by the rubbing of our clothing. If one remove the thin and apparently hairless skin of the anterior aspect of the arm and transplant it to the face, it, no longer repressed by the friction of the clothing, will develop a magnificent growth of hair and the patient will be obliged to constantly pull them out. This was exemplified in a case operated on by the speaker for the restoration of the lower cul-de-sac. It is now hoped that the artificial eye, the patient will soon wear, will keep the hair down. In another case where an angioma the size of a butternut was exercised from the lower lid of a child 2 years old, the skin removed at that time involved the whole length of the lower lid, and to replace it a Wolfe graft was used. It took nicely, but now there is a large, white, thick patch, resulting from the transplantation, to deal with, however successful it has been and however useful it is. The speaker in nearly all cases discards the Wolfe graft and advises the Thiersch, which an-

swers all requirements and avoids the inelegant appearance following the thicker grafts from whatever part of the body they are taken.

DR. OSCAR DODD, Chicago.—I was very much pleased to hear this paper by Dr. Hotz, and especially the suggestions as to the anchorage of the grafts of the lower lid to prevent retraction. In the upper lid I have never had any trouble in using Thiersch grafts, and that is the only form I use in that region, unless the whole thickness of the lid, with the conjunctiva, is removed with the tumor. In the lower lid I am not always able to get tissue of the size that can be transplanted. In two of my cases the burn was so deep, there was nothing but dense cicatricial tissue adherent to the periosteum, and all I could do was to bring tissue from outside to form the lid. Thiersch grafts I have found useless in these cases. Perhaps by anchoring, the method Dr. Hotz describes, I may be able to obviate the difficulty. The Wolfe graft, if prepared very thin, I have found of benefit, but it has the disadvantage that it is never like the skin of the face or lid, and will remain—at least, for years—of a different color and can be distinctly seen. As for pedicle grafts from the temple and cheek, I do not feel as Dr. Hotz has stated, that they should never be used. I think there are cases where it is impossible to get as good results as we wish in the restoration of the lids without them.

DR. GEO. F. SUKER, Chicago.—Dr. Hotz's operation is certainly admirable. He laid emphasis on the contraction of the flaps not taking place, which is the all important factor. It occurred to me, however, that if he did not have the exact coaptation by not having the upper flap overlap the lower, but left a narrow line of granulating surface between the flaps, he would avoid all depression or contraction. I agree as to the Thiersch graft. I had a disagreeable experience not long ago in transplanting a Wolfe graft, as now the patient has to shave his lid.

DR. DERRICK T. VAIL, Cincinnati.—This is a subject which appeals to us all. We have seen the various forms of ectropium due to cicatricial changes following burns and neoplasms about the eyelids. The ingenious method of Dr. Hotz certainly will cover many of the cases we have to treat. I wish to compliment

him on his ingenuity and originality in devising the double flap and wish to inquire if it is original.

DR. HOTZ.—So far as I know.

DR. VAIL.—It is a valuable contribution to the art of plastic surgery of the eyelids and I think will prove to be of great help in the surgical treatment of cicatricial ectropium. I have never used it, but have used his old operation for ectropium with fairly satisfactory results.

There is one kind of ectropium of which I have seen two cases within eighteen months, where his double flap operation would not, perhaps, be applicable. I refer to that form of ectropium which involves the inner canthus of both eyes; where the skin of the nose has been burned off, followed by cicatricial contraction and the inner canthus of each eye is drawn so badly toward the median line of the nose that there is only a small space between them on the bridge of the nose. This generally concerns both eyes. I met this condition with the suggestions laid down by Dr. Hotz in his former operation. I restored the lids to the eyeball and also restored the patulency of the lachrymal ducts, laying in a nice transplanted flap, but the operation was a failure. The cause of the failure I learned by the experience afforded by the operation. In restoring the lids to the eyeball at the inner canthus, you have a crescentic exposure of the fascia, and it would seem that a piece of skin laid in there would give satisfactory results, but it will not. To have a good result it is necessary to anchor the upper lid to the lower lid at the inner corner of the eye in such a way that you really have an involution of the canaliculi and puncta to allow for contraction changes. The main thing about the whole operation for cicatricial ectropium in general is in following the suggestion Dr. Hotz has made about *taking up the slack of the lid* and unless you take away fully a third of the lower lid at the outer canthus your operation will be a failure.

DR. ALBERT E. BULSON, JR., Fort Wayne, Indiana.—It is my experience that the annoyance from the presence of hairs in the skin grafts, already mentioned, may in a very large measure be overcome by taking the grafts from behind the ear where the epithelium contains fewer hair follicles. These grafts prove more satisfactory than grafts taken from any other part of the body, but it is not always possible to secure grafts of sufficient

size to cover large surfaces and then resort must be had to epithelium from some other part. In the majority of operations around the eye, grafts of sufficient size, taken from behind the ear, can usually be obtained.

DR. J. M. RAY, Louisville, Ky.—I presume all the members of this Association listen with interest to what Dr. Hotz may say about the plastic surgery of the eye and eyelid. I have done a certain amount of this work myself and I find it a great advantage, when I transplant flaps, to first bare the edges of the lids and sew them together. I did this in one case with a large Wolfe flap, a very small portion of the flap grew, but by having the eyelids sewed together, I later transplated Thiersch grafts and eventually covered the surface. I then separated the lids and a good result followed.

DR. HOTZ (closing discussion).—I was glad to hear Dr. Wood's observations. Whether I am correct or not, I emphasize the careful selection of the proper materials. (Dr. Hotz showed photographs of cases operated on by other methods with unsatisfactory results.) Cut Thiersch grafts as thin as possible—just the epidermis—just deep enough to get live cells and not hair follicles. Do not transplant hair follicles and you will not have hairs in the flaps. If in the lower lid cicatricial tissue is not available, I would use Thiersch grafts. I have done the operation a number of times, otherwise I would not present it to you. I could show you in Chicago a number of cases. The upper lid moves nicely and shows the deep characteristic furrow. Dr. Suker seems to have misunderstood what I said in regard to allowing the upper flap to overlap. This is done to prevent the rolling in of the flaps, as Thiersch flaps have a tendency to roll in. So fastened, they will not do it. In changing the bandage, the overlapping portion is simply pared off. I forgot to mention that. To leave a granulation line along here would have no advantage. If we fasten the flap firmly to the tarsus we know it will unite. If we leave it to granulations, we trust to uncertainties.

(Just at this time a patient, whom Dr. Hotz had formerly operated on, chanced to call, and thus the Doctor was afforded an opportunity to present the case for inspection to the Academy and to show the perfect cosmetic result obtained by his operation).

EPISCLERITIS AND SCLERITIS.

BY ADOLF ALT, M.D.

WHEN deciding to bring before this Association the subject of episcleritis and scleritis, I was aware of the fact that it is not a very enticing subject. These diseases belong to the class of which we know as yet but very little outside of their clinical picture, and in which, in consequence, our therapeutic efforts are chiefly empirical, and, though sometimes crowned by apparent success, are in the majority of the cases utterly unreliable, not to say useless; perhaps, sometimes even harmful.

I was also aware of the fact that I could not add anything very new or striking to the knowledge of these diseases, but I hoped that by treating the subject more or less in the light of the results of my own experiences with it, I might induce others to detail theirs, and that, perhaps, after all, this sterile field might be rendered fruit-bearing.

When looking through the many and more and more swiftly multiplying text-books it seems that one copies from the other with little or no addition from personal experience, especially as concerns the subject of this paper, and our knowledge concerning it has of late not been enhanced to any appreciable extent.

The clinical picture of episcleritis and scleritis is so well known to all of you that I need not take up too much time in detailing it.

We may clinically differentiate between superficial episcleritis and scleritis, and a more deeply seated scleritis.

In the superficial form one or several foci of inflammation are visible under the bulbar conjunctiva. These form characteristic roundish red, or reddish-blue elevations, covered by congested conjunctiva. This conjunctival and episcleral congestion may be confined to the seat of the swelling and a few vessels supplying its area, or it may be more general in character. The elevated nodule is in most cases more or less solid and feels rather hard to the touch, but I have also seen a number of cases in which during the progress of the disease elevated spots were formed which looked exactly like large phlyctæna, semitransparent and as if filled with a muddy yellowish liquid. The nodules cannot be moved on

the sclerotic. Pressure on them, which is usually quite and sometimes extremely painful, may empty the superficial conjunctival blood-vessels, but not those lying in the depth, nor reduce the swelling.

Such a nodule may disappear and consecutively more nodules may spring up, and it may happen that this nodule-formation travels gradually all around the periphery of the cornea.

The seat of the nodules is either quite close to or but little removed from the periphery of the cornea, or farther back near the aequator of the eyeball. In a general way, though not without exception, their seat seems to be near the places where larger blood-vessels pierce the sclerotic.

When the nodules are formed very close to the periphery of the cornea this membrane is frequently seen to suffer also. I have seen typical marginal ulcers of the cornea thus formed corresponding to the seat of the episcleritic nodule. In other cases, and this happens more frequently, an infiltration into the deeper corneal tissue takes place, which gradually leads to the formation of new tissue, and consequent sclerosis of a localized peripheral part of the cornea.

In some cases the eruption causes little or no discomfort, even though it gradually invade quite a large area of tissue. In most cases, however, there is spontaneous pain and great discomfort and the eyes cannot be used for any work.

This is the more unfortunate since the disease, in the majority of cases, attacks both eyes, although I have seen it in quite a number of cases to remain confined to one eye only.

During its springing up, growth and disappearance such a nodule never shows any signs of superficial or deeper necrosis and no ulcer results from it. When the affection heals, with or without treatment, the swelling becomes gradually reduced in size, till it finally disappears, and usually together with it the congestion which had accompanied it. In other cases the congestion remains behind and new abortive attacks can still be recognized by a sudden localized increase of congestion, although no swelling appears, till finally, like distant lightning after the storm, even this fades out.

The seat of a former episcleritic nodule may then show no trace of having been disturbed. In most cases, however, and

especially in the more prolonged ones and those in which the nodule was situated near the aequator, a grayish-blue or grayish-violet spot marks the site of the former infiltration for life.

The course of the disease is pre-eminently slow, and with the continually renewed eruptions it may cover many months and years. I have at this time a patient under my observation, in whom the disease made its first appearance nineteen months ago, and at this date is far from being cured.

The disease is usually seen in adults, yet sometimes it also appears in young children. It seems to attack individuals of the female sex oftener than those of the male sex. In some cases I have noted a decided influence of the female sexual sphere on the course of the disease, as it showed exacerbations with every menstrual period. Negroes are often subject to it.

The clinical picture of the deeper-seated scleritis is a different and more varied one, according to the area involved and its complications, and its sequels are of a much more deleterious character. It has always seemed to me that this is an essentially different affection, although its symptoms in certain cases and at a certain period may in a measure be similar to those of superficial episcleritis. When the deeper parts of the sclerotic in the anterior half of the eyeball are inflamed we may, also, see an isolated elevation of the sclerotic of bluish-red tint, covered by congested conjunctiva. This elevation usually covers a larger area than in episcleritis. It is in some cases seen to form a bluish elevated ring around the periphery of the cornea, close to it or more over the region of the ciliary body. When the disease, as it undoubtedly does, attacks the sclerotic farther back and near the posterior pole of the eyeball, we can, of course, not see it.

Such deeper seated scleral infiltrations may also gradually disappear like the superficial ones, without leaving a mark behind. Yet the rule is that when such a nodule has disappeared we find in its stead a bluish-gray thin area in the sclerotic, to which the uveal pigment gives a peculiar color. This thinned out area is too weak to withstand the normal, and much less an increased, intraocular pressure and the result is a more or less localized staphyloma. If the disease

had attacked the circumcorneal tissue this whole area becomes stretched and the result is the annular staphyloma which used to be termed "Intercalar Staphyloma." In the same manner ciliary and aequatorial staphylomata, as well as more posteriorly situated ones, and a total scleral staphyloma may result.

This form of scleritis is in my experience always combined with a more localized or general affection of the uveal tract. While the question remains which of the two membranes is the first to be attacked, my opinion is that the uveal tract is, as a rule, first diseased and the sclerotic secondarily.

Deep-seated scleritis is more frequently observed in very young children, although it may also be met with in adults.

The course of the disease with its sequelæ is much longer than that of the superficial episcleritis.

Fuchs (and before him some English authors) has described a third form of episcleritis, to which he gave the name of episcleritis periodica fugax. The symptoms of inflammation in this form are said to be slight and of short duration, although recurring again and again, so that this affection also may extend over years. It is said to be a very rare form, and I have never seen it.

If we now inquire into the etiology of these affections we find an astonishing unanimity among the authors of textbooks. A rheumatic diathesis always heads the list of etiological factors, and some authors go so far as to state that they have hardly ever seen a case of episcleritis that could not be explained by a rheumatic diathesis. I must differ very materially from these authors. In my experience cases of episcleritis in which a history of former rheumatism in whatever form could be elucidated, or in which a rheumatic diathesis was present at the time, have been but very few indeed. But I confess I am in decided need of information as to what the authors exactly mean by rheumatic diathesis.

Next to rheumatism a gouty diathesis is most frequently accused. A gouty or uric acid diathesis has of late attained such a prominence in the explanation of all sorts of affections that it is not to be wondered at that it may also cause episcleritis. I have signally failed in my cases to convince myself of any gouty diathesis, and while I know a great many

individuals who are undoubtedly afflicted with the so-called uric acid diathesis, not one of them has ever, so far as I am aware, suffered from episcleritis.

That acquired syphilis may once in a while cause a typical episcleritis has been proven by Alexander not only from his own observation but also from previous literature (Coccius, Jacobson, Arlt, Mooren, Galezowski and Higgens).

Tuberculosis and scrofulosis are also mentioned, as is hereditary syphilis. Perhaps these affections play as important a rôle, or even a more important rôle in the etiology of at least the deep scleritis, if not the superficial one, than

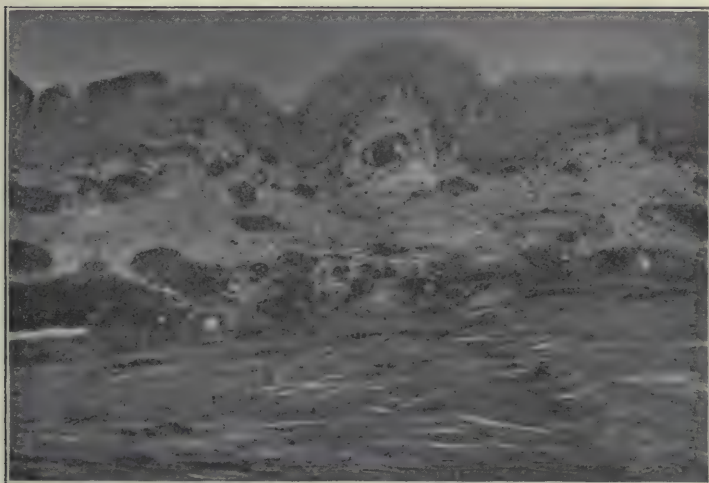


FIG. 1.

either rheumatism or gout. This may be, and I think it is, especially so when the disease is found in young children.

Still, in summing up my clinical experience with these affections of the episcleral and scleral tissue, I must confess that in most of the cases I have been utterly unable to convince myself of a general diathesis which might underlie this local affection, even though certain so-called specific remedies seemed to, and for the time being did, exert a beneficial influence on its course, and the diagnosis of the diathesis might have appeared as proven *ex juvantibus*.

In this general uncertainty we might hope to gain some special insight into the mysteries of the affections we are here considering by the study of their pathology. Yet, eyes with

superficial episcleritis have, from the nature of the disease, but very rarely and only, so to speak, by accident reached the pathological laboratory, those with a deeper-seated scleritis, while much more frequent, are usually seen when the most active stage has long been passed and only its sequelæ can be studied.

The following is a resumé of what has been seen: Superficial episcleritis and scleritis (because the two are always found together) has been studied especially by Uhthoff and Schirmer, and I have examined one case. In my case the affection was strictly a superficial episcleritis and the swelling was situated near the cornea. The conjunctival epith-

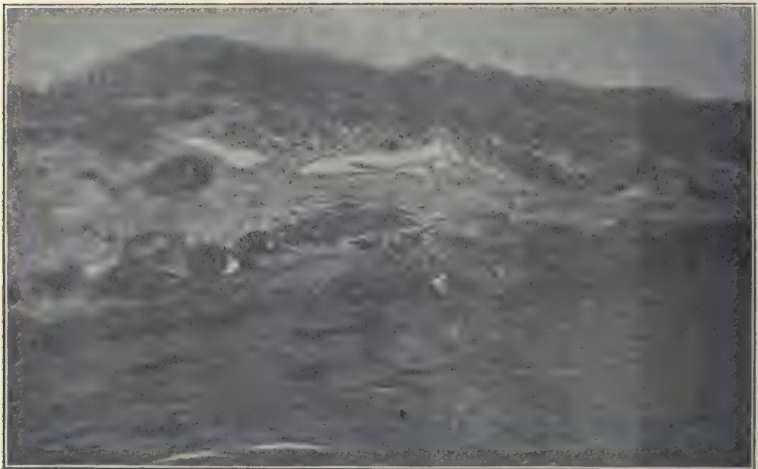


FIG. 2.

elium is thickened. Underneath it lie the greatly congested conjunctival blood-vessels surrounded by varying masses of round cells. The conjunctival and episcleral tissue is somewhat œdematous and its elements are pressed apart. In it are seen a number of evidently enlarged lymph-vessels, and their number appears greater than in the norm. The episcleral blood-vessels are enormously engorged, so that they form almost a continuous layer, the small interstices of which are filled with round cells. The round cell infiltration reaches also into the superficial layers of the sclerotic. (Figures 1, 2 and 3.)

What I find in this case agrees perfectly with what

Uhthoff and Schirmer have seen, only in their cases the round cell infiltration seems to have reached a little deeper into the scleral tissue. Uhthoff, moreover, in his specimen found evidences of small hæmorrhages which are very likely to happen in a tissue so gorged with blood. The normal elements of the tissues did not seem to be materially altered in any of these cases.

Cases of deeper scleritis at a florid stage have been described by Schirmer, Uhthoff, Holthouse and Collins, Schlodt-

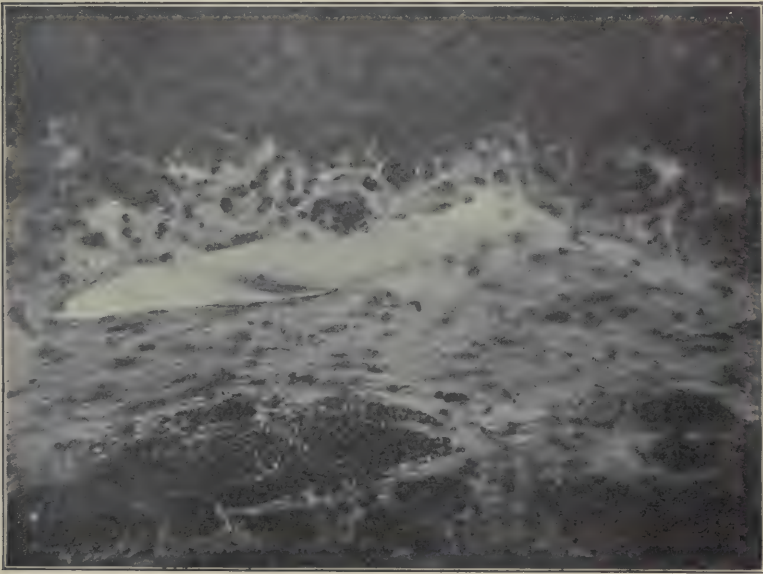


FIG. 3.

mann, Friedland, Kostenitsch and Ginsberg. I have seen a number of them also. While in these cases the episcleral tissue is always involved in the process of inflammation, it is especially the scleral tissue which suffers. There is usually a very considerable infiltration of round cells in the superficial layers of the sclerotic fibres which quite frequently assumes the shape of tubercle-like round and oblong nodular formations. The pressure leads to hyaline degeneration or necrosis of the scleral fibres. Friedland found, also, some giant cells. The pressure of the increasing number of round cells may also lead to necrosis in the round cell accumulations themselves, so that they lose their cellular characteristics

and form a more uniform semi-hyaline mass. (Figure 4). The underlying part of the uveal tract is usually also inflamed. Later on, when the inflammatory process has subsided, the sclerotic is thin, there are few cells visible in it, the blood-vessels are obliterated and the corresponding part of the uveal tract is also atrophic and attached to the sclerotic.

When the scleritis is still more virulent, we find also the inner layers of the sclerotic and the corresponding part of the uveal tract infiltrated with round cells, so that the boundary line between the two membranes cannot be distinguished. Between the infiltrated superficial and deep layers of the

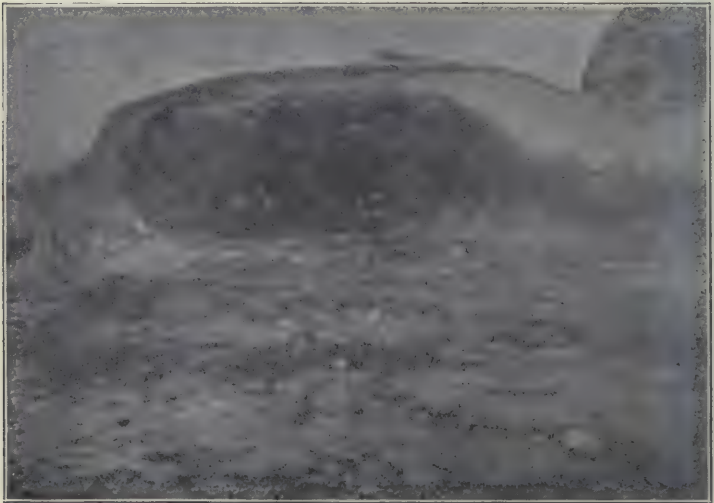


FIG. 4.

sclerotic there is usually a middle part which seems but little affected. In other cases the infiltration concerns all the layers of the sclerotic. All episcleral and conjunctival blood-vessels are gorged with blood and the lymph-vessels appear to be much more numerous and larger than in the norm. Necrosis of the scleral fibres and of the infiltrating cells is usually seen in these cases.

The process leads finally to atrophy and stretching of the affected parts (staphyloma), yet it may also, and it does so usually in the parts near the posterior pole of the eyeball, lead to the new formation of connective tissue and thus cause a so-called hypertrophy of the sclerotic, as we are accustomed to find it in phthisical eyeballs.

This in the main is the description which is given by the different authors and also of my own observations. I might add that the deep scleritis, like the superficial, is found as a rule where the larger blood-vessels pierce the sclerotic, and I am of the opinion that this form of scleritis is an affection secondary to an affection of some part of the uveal tract. The most undoubted example of this we see in cases of florid panophthalmitis in which even the orbital tissues surrounding the eyeball may be drawn into the process of inflammation.

Herein also lies the reason that I believe that the superficial episcleritis is a disease *per se* and distinct from the deeper scleritis. I further believe that Schlodtmann is right in assuming that episcleritis is probably a special mycotic disease, the parasite of which, as yet unknown, may some day be found.

Although giant cells have been found in some cases of deeper-seated scleritis, no bacilli of tuberculosis have been demonstrated. Neither could syphilis be assumed to explain the histological findings.

Pathological research, as is seen from the foregoing, has thus far not succeeded in throwing much light on the nature and etiology of the diseases under consideration. Yet, clinically they form a distinct group which is not to be confounded with other affections. It is, therefore, clear that, perhaps, a great many more such cases will have to be studied pathologically, from a histological as well as a bacteriological standpoint, in order to solve their mystery.

Here, maybe, you will permit me to swerve a little from the subject of this paper and put in a plea for the pathologist who, if ever so willing to do the work, cannot get the necessary material for study, unless it is supplied to him by those of his colleagues who do not themselves make use of it for special study. There are still too many interesting and important eye specimens simply cut to pieces, or allowed to rot and dry up in jars, when in the proper hands they might help to solve one or the other question of importance to science—that is to humanity. No worker in this field can get enough material from his own practice. What a wealth of material might he have to work on if no specimen was thought too trivial to be submitted to his study!

After this *oratio pro domo* let us return to our subject, as we have still to consider the treatment of episcleritis and scleritis.

In correspondence with our lack of knowledge as regards the cause and true character of these affections, we find a perfect wealth of recommendations in the text-books. As most, in fact all, of them consider rheumatism and gout the prime causes of episcleritis, they are unanimous in giving first place in its treatment to anti-rheumatic remedies, especially the salicylates, and of late with preference aspirine. It has also been my experience that these remedies seem to have a beneficial influence in some cases, at least as far as relieving the pain and discomfort are concerned. Yet they will do the same in non-rheumatic affections, as, for instance, in syphilitic iritis. I have tried them faithfully and sometimes, perhaps, only too persistently, in episcleritis, and I have convinced myself time and again that, as far as curing the disease is concerned, they are unreliable. I must say the same of the iodides and mercury internally, except in syphilitics. Subconjunctival injections of solutions of different mercurial salts and of pure salt solution have sometimes a beneficial action, but in other cases they seem to add decidedly to the irritation present and to aggravate the disease. Pilocarpine locally, internally, or subcutaneously does sometimes well, so do heat and cold, but their efficacy seems to give out after a little while, like that of the other remedies previously mentioned, whether we combine with them mydriasis and rest in a dark room, or not. Massage with yellow oxide of mercury, aristol or salicylic acid, in the form of ointments, is decidedly beneficial up to a certain point and then disappointing. I have never practiced the removal of the episcleral tumor with the knife, cautery, or sharp spoon, as has been so highly recommended by some, nor peritomy of the conjunctiva.

One remedy I have always at first tried in the last ten years, and that is the inspergation of calomel. With it, especially in recent cases, I have had a number of excellent results. In fact, having had a series of cases which in comparison to other methods had yielded very promptly to this remedy, was what prompted me to bring this subject before you. I had even thought that I was the first to use it and

to recognize its value in this affection, when, as is so often the case, I found this was a mistake and that Nettleship, also, recommended it. I raise no question of priority. But, as I said, I was almost convinced that I had found a reliable remedy for the disease and was highly elated, when two cases followed in rapid succession in one of which its action was neutral and in the other decidedly bad. In spite of this I feel that I should advise you to give calomel a fair trial. Most recently adrenaline and all the other derivatives of the suprarenal gland have been recommended for this as for a good many other eye diseases. I have tried them, also, and found them wanting. Brilliant adjuncts as these remedies are, I have still to see the first case in which they have a lasting therapeutic effect. To be sure, with them you can blanch the affected area, but their therapeutic effect is nil.

If we are at a loss what to do in the treatment of the superficial episcleritis, this is even more so in cases of the deep seated scleritis. In these the complications must have a decided influence on our therapeutic measures and it would lead us too far, would we here consider all the different possibilities. However, I may say that mercurials have now and then a good effect, as we might expect from our experience with these remedies in cases of disease of the uveal tract.

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DISCUSSION.

DR. D. S. REYNOLDS, Louisville, Ky.—I think one of the unfortunate things in considering this subject is the nomenclature. "Episcleritis" does not mean anything except inflammation on top of the sclera. "Epi" means upon. Episcleritis would necessarily mean something external to the sclera. I watched closely the description and Dr. Alt describes

scleritis all the time, and if I caught his meaning, I would say it is the opinion of the writer that scleritis nearly always begins within the walls of the sclera. As the sclera is not disposed in layers, I cannot conceive of a superficial or deep-seated scleritis. It is made up mainly of heterogenous connective tissue fibres, and at the points where the wall is perforated by nerves and blood vessels the nodular masses described by Dr. Alt occur. When you study the course of syphilitic disease in its remote manifestations—that is to say, inherited syphilis—it will be observed that the circumscribed organic structural changes accompany the course of the blood vessels. They have their origin in those parts where there is strong resisting tissue. Here is most likely to be the seat of gummatous deposits. I think a large proportion of the cases are inherited syphilis, beginning as a gummatous formation on the uveal surface. I have a large mass of clinical notes on these cases, and I cannot recall from memory an instance that was not seen in a syphilitic subject. I have not seen it in acquired syphilis, but in children the subjects of inherited syphilis, innumerable times. I have had the same experience in the treatment of scleritis as in the treatment of inherited syphilis in other parts of the body. I employ mercury in small doses, giving strict attention to diet and habits of life, with the design of improving the general condition of the patient. I believe that a great deal depends upon nutritious foods, and the use of mercury in small doses will terminate nearly all cases of scleritis.

DR. J. O. STILLSON, Indianapolis.—This is a very interesting subject and I can but express my high appreciation of Dr. Alt's admirable paper. I feel when I hear him read a paper—and I feel it more than ever this morning—under obligations to him for the clear and charming manner in which he gives us the picture of this most distressing and unsatisfactory disease. We have all met this disease in the course of our practice, I dare say, and if the experience of the other gentlemen has been anything like my own, I am sure they can bear Dr. Alt out in this seeming quandary as to what the disease really is and what really is the rational method of treatment. It will not do for us to start out upon the gouty and rheumatic diathesis, as we are so prone to do, to clear up the diagnosis, to understand this disease, because that standpoint

will fit too many other obscure cases. It will not do for us to classify this as a *terra incognita*, because we are too well acquainted with the land marks and the tissues here. It will not do for us to hold up our hands and declare it is incurable. Eighty per cent of the people have gout or rheumatism, that is, are of a lithic diathesis. Eighty per cent of the people who live to be along towards the fifties have rheumatism, and if we are going to take that as a basis, we can make up our minds we can explain anything on the uric acid diagnosis. It is like the old explanation of glaucoma: we can easily explain glaucoma if we want to take the backward track. But when we come down to the fact that the majority of men (about three-fifths) are hypermetropic anyway, then it will not do to use hypermetropia as a cause of glaucoma. We see it more in hypermetropic people because there are more hypermetropic people. And so, too, on this line, we may well say that we see more episcleritis in rheumatic or gouty people than in those free from this disease, because more people have this disease than do not. So to my mind it is a co-incidence rather than a cause. I do not agree with Dr. Reynolds about it being in so many instances a specific disease. I used to feel that way myself, but I know that I have seen cases where this could not be accepted. I studied a case about a year ago in connection with Dr. Stevenson of Ft. Wayne, in which we found nothing that would lead me to believe it was syphilitic.

DR. J. A. DONOVAN, Butte, Mont.—I have had quite a number of cases of this disease and never have been able to connect it with syphilis either directly or indirectly, and I look for it in my patients. Some time ago I had two patients at the same time who had a peculiar odor to the breath in a warm room, which struck me as peculiar. I noticed the same odor on another patient, and ever since I have made the diagnosis from the characteristic odor of the patient. The last half dozen have had a peculiar odor perceptible. The last I had was a lady who had a history of having been treated for three years by local treatment. I began with one-fourth grain calomel until she had taken three grains every day, and after that phosphate of soda. These are the only patients with whom I have had satisfactory results in this trouble, and so far every one has recovered within five to six weeks.

DR. GEO. F. SUKER, Chicago.—I think this is a very opportune time to discuss episcleritis and scleritis. I am sorry Dr. Alt did not say anything definite regarding sodium salicylate. I have several cases at the present time in which part of the cornea is involved, and I agree that we usually have an inflammation of the uveal tract. I have a patient who has had episcleritis for a year, with the characteristic bluish color, and nothing relieved her. As a last resort I started her on the salicylates; and as I am a firm believer in large doses, I ran up to 150 to 180 grains per day. Her pain subsided and she is recovering nicely. She is still taking 60 grains per day with plenty of water. I make the patients take as much water as the skin can hold while taking these large doses. In addition to these large doses, I use the atropine and hot fomentations. This person is making a very fair recovery; the nodules are disappearing, but the bluish cast remains. As to the staphylomatous nodules about the eye, I fully agree with the doctor. I saw one case quite recently with Dr. Beck which has now developed not only a staphyloma but also a glaucoma. She had terrific pains and nothing did any good until she had the large doses of the salicylates. As to the etiology, whether rheumatic or syphilitic, I agree that we can't tell what it is. Some seem to be one and some the other. If you give large doses of sodium salicylate you can possibly exclude a syphilitic basis. Still even in specific cases it is efficacious. Whether the uveal tract is the basis or not, I think Dr. Alt will clear this up for us some time.

DR. C. L. MINOR, Springfield, O.—Recently two young ladies, aged respectively 18 and 20, came to my office with involvement of the sclera. I referred them to the family physician for examination. There was no history of syphilis in either family and no gouty diathesis that could be found. They both had some derangement of the female organs which required operation for correction and soon after these operations the scleritis disappeared and up to the present time has remained cured.

DR. J. E. COLBURN, Chicago.—I would like to offer one suggestion regarding the treatment, and that is the climatic. I have had a few cases and have noticed that the most severe exacerbations usually came, as in choroiditis, in the spring, beginning in March and continuing during April and May.

One patient invariably had a return of the attack during these months, and at my suggestion she went to California about mid-winter four years ago and escaped the recurrence. An opportunity occurred for her to go to Hawaii, where she has remained for three years. In a letter from her recently she said she had no return of the conditions. She had the colored spots on the sclera and I had her under observation for a number of years. I tried everything ever recommended for this disease before suggesting climatic change.

DR. ALT (closing discussion).—I perfectly agree with Dr. Reynolds as to the meaning of the word “epi,” but it has been the custom to call the tissue which lies upon the sclera and connects the conjunctival tissue with it, the episcleral tissue. As I have described, and as you see in the photographs, in episcleritis the inflammation lies in this tissue which unites the two membranes together and therefore it is right to call it an episcleritis. In these cases there is no inflammation to be found in the uveal tract. As regards syphilis being the underlying cause in most cases, I may ask in what disease may we not find some form of syphilis present? How can you prove it and how can you disprove it? Dr. Donovan must have a good sense of smell if he can diagnose scleritis from the odor. At any rate, as he stated that he has been successful with phosphate of sodium, we should give it a trial. Dr. Suker said I had not mentioned salicylates. He was evidently not present when I read the paper. I am sorry that in the whole discussion I have heard only repetition of what I have said, excepting from Dr. Donovan. No one seems to have better success than I, yet let us hope that the future will bring it. Dr. Minor mentioned the female sexual organs as possibly having some effect. I also mentioned this and said that in one case exacerbations took place with each menstrual period. I also send my patients to the general practitioner whenever the eye trouble seems to be connected with some general disease and have them thoroughly examined. With regard to the climatological treatment, I have no experience, but it is interesting to hear Dr. Colburn’s statement. It may be that the hot climate in those countries acts as a beneficial factor. Heat or sweating in some cases are very beneficial.

EXSECTION OF THE SO-CALLED TARSAL CARTILAGE IN CASES OF CHRONIC TRACHOMA.

By CASEY A. WOOD, M.D.

CHICAGO.

Illustrated with 8 Cuts.

SOME five years ago* I reported the results of the so-called Heisrath's operation in fourteen cases of chronic trachoma. As my subsequent experience has been equally and uniformly satisfactory, I am encouraged to bring the subject once more to the notice of the profession through this Association, particularly as I do not believe it has received the attention it deserves.

Let me repeat that this procedure is not, in my opinion, indicated in any of the recent or acute forms of trachoma nor would I advise it in any case where there is a reasonable prospect of early cure—not temporary relief merely—from any other form of treatment. When other remedies have failed and there is nothing before the patient but months or years of suffering—nothing but the “ups and downs” that characterize a deepseated trachoma, with its visual dangers and bodily discomforts from pannus, corneal ulcer, trichiasis and entropion, not to mention long continued and serious interruption of work—in many of such cases tarsal excision is certainly indicated. On the other hand, in the most advanced stage of the disease, in those cicatricial forms that have gone on to shrinking of the sac and in which there are probably few or no active trachomatous nodules in the tarsal cartilage, I do not think the operation is justifiable. Nor should it be resorted to if it is possible, as Gifford points out that it sometimes is, to remove, one by one, the discrete and scattered trachoma nodules from the tarsus itself.

Let me further declare myself in this important particular by more positive statements. Removal of the tarsus, in part or as a whole, is indicated in those long standing cases of trachoma, not amenable to other forms of treatment, in which the lids show trachomatous infiltration, with granulation deposits in the connective tissue of the retrotarsal folds, whether the cornea be affected or not. If to these conditions

*Removal of the Tarsus and Retrotarsal folds in certain cases of chronic Trachoma. *Annals of Ophthalmology*, 1898. p. 372.

be added thickening and enlargement of the tarsus itself, the operation is even more urgently indicated. Also, when there is evident disease of the folds, without apparent thickening of the cartilage, but the cornea is implicated, the operation should be done. A very important class of cases, from an operative standpoint, is that where with atrophy or cure of previously existing granulations in the tarsal folds there remain deepseated foci in the tarsus. In this troublesome and inveterate form of trachoma, whether the cornea has escaped or not, removal of the tarsus will give gratifying results.

The palpebral conjunctiva is rarely the only site of granular deposits in long standing cases of the disease. It is quite exceptional that the tarsus and submucous connective tissue escape. I believe that I am correct, therefore, in asserting that the simple method of excising the retrotarsal folds (long ago advocated by Richet and Galezowski) does not meet the requirements in such cases. The proposition is practically to remove the neoplasms that, in the later stages of this infection, are responsible for the destructive lesions of the disease. Why, then, should we remove a portion of these semi-malignant tumors and allow the others to remain? As long as there is reasonable ground for assuming that the activity of the trachoma colonies is confined to the conjunctiva and submucosa such procedures as *grattage*, the use of forceps, cauterization, excision of portions of diseased membrane, etc., are of course, proper, and the method that I am about to describe is not intended for their relief.

Although it is desirable that the eye should be as quiet as possible before operation, I have not hesitated to excise the tarsus either in the presence of corneal ulcer, increasing pannus or during an acute exacerbation of the chronic disease. I have been satisfied with the results in these instances but have been careful to remove the stitches at as early a date as possible and to keep up constant disinfection of the eye while they are *in situ*.

A better understanding of the effects of the operation is gained by a reference to the muscular supply of the lids. According to Thomas Dwight and others the superior rectus, besides its insertion into the globe sends fibres not only to the top of the fold of conjunctiva, which is thus pulled up and back

in harmony with the upward excursion of the eye but also to the top of the tarsus. Moreover "the levator broadens out into an expansion stretching across the whole orbit from one bony wall to the other, which, by its outer portion separates the

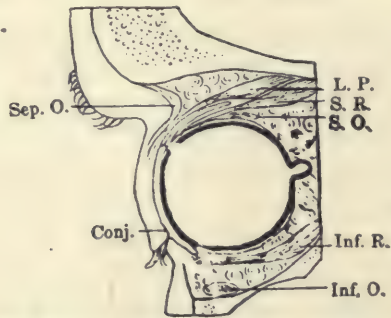


FIG. 1—(Dwight). Showing the relations of the orbital fascia to the tendons of the levator palpebrae, the superior rectus and the tarsus.

greater lachrymal gland from the accessory portion below it. This expansion splits into two layers. The greater portion, consisting of involuntary muscular fibres, (Mueller's muscle), is inserted into the upper portion of the tarsus, while certain anterior fibres pass into or through the fibres of the orbicularis to the skin of the lid. Their function is to draw the skin to the

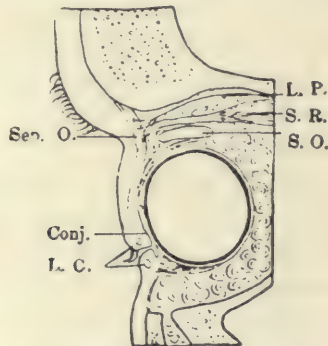


FIG. 2—From Dwight, showing the relations of the orbital fascia to the upper lid.

fold above the tarsus when the lids are opened. The expansion of the levator passing to the tarsus consists largely of unstripped muscular fibres mingled with elastic tissue. This is connected with other involuntary fibres arranged transversely, the whole constituting what is known as "Mueller's muscle."*

*Norris and Oliver. System of Diseases of the Eye. 1. 91. 92.

lower lid, the tendinous expansion of the inferior rectus dividing into three layers, one of which is attached to the tarsus and taking the place of the levator of the upper lid in drawing the lid down in downward rotations of the globe and in opening the eye.

All the German authorities consider cocain a sufficient anesthetic for the operation. Kuhnt instills a 4 to 10 per cent. solution as a preliminary and then makes two or three subconjunctival injections of a 6 to 10 per cent. solution. I have not found this very satisfactory with my patients and I now invariably insist upon a general anesthetic. Not only is the operation a painful one (particularly when more than one



EXCISION OF THE TARSUS.

FIG. 3.—First act. Eversion of the upper lid with forceps (Kuhnt).

lid is involved) but its success largely depends upon precision in placing the sutures and in other details difficult to carry out if the patient be restless or nervous.

The diagrams, from Kuhnt's work, illustrate the various steps of the operation, which I shall now proceed to describe.

When the operation is done, as it usually is, on the upper lid, the latter is everted so that the convex border of the tarsus is thoroughly exposed. This is now firmly grasped by two strong, toothed forceps at the junction of the middle with the outer and inner thirds of the tarsal margin, and drawn firmly upward by the assistant standing at the patient's head. The junction of the palpebral and ocular conjunctivæ is now fully exposed and may be readily examined. Following as nearly as possible the margin of the diseased area, an

incision is made from the outer to the inner canthus through the conjunctiva only. Unless, in consequence of previous mechanical treatment, the conjunctiva is bound down to the



FIG. 4—Second act. Complete eversion of the upper lid and exposure of the retrotarsal folds. First incision along the dotted lines (Kuhnt).

underlying tissues, the wound will gape and the fibres of Mueller's muscle may be recognized. Three stitches should now be passed through the bulbar margin of the incision, care being taken to include only the conjunctiva and a few



FIG. 5—Third act. Second incision near the lid margin, after placing of the sutures in the upper border of the first wound (Kuhnt).

fibres of the submucosa. If more than a mm. in width of conjunctiva is included in the sutures, small symblepharon folds may form opposite each stitch, and if too deeply inserted there will be a noticeable dragging on the lid edges, as occurred in one of my own early attempts.

A word as to the sutures. My assistant, Dr. Frank Brawley, has prepared for me a modification of the black silk (preferably No. 2 black braided) which Worth advises in his advancement operations. I have used them for the past year with great satisfaction in all operations that involve the conjunctiva and I warmly advocate their employment in the procedure about to be described. The silk is first wound upon ordinary glass microscopic slides (for convenience of handling) and sterilized by boiling 30 minutes. It is then dehydrated by immersion in absolute alcohol for 10 minutes and the drying process assisted by holding the slides a few feet above a Bunsen burner flame for a few additional minutes. The slides of silk are then dropped into a jar of paraffin con-



FIG. 6—Fourth act. Bringing the edge of the wound together after tarsal excision. Estimating the proper place to enter the needles below.
Forceps used in operation (Kuhnt).

taining 25 per cent. of vaseline, where they remain until used. Each time they are used the jar containing the silk is resterilized by heating, an end of suture is drawn out of the jar and the excess of wax is "stripped" off the required suture lengths by drawing it through sterile gauze held between the thumb and finger. The threads are now somewhat stiff yet flexible, are easily threaded, never "kink" and slip through the tissues with the minimum amount of friction and traumatism and do not readily tear out of the tissues in which they are placed. Moreover, knots made in these threads are much less likely to irritate and abrade the cornea or bulbar conjunctiva. Once introduced through the lower wound margin they should be allowed to hang down over the globe (see the diagram) and to rest on a sterilized towel placed on

the cheek. After the sutures have been thus placed the bulbar conjunctiva should be separated from the globe a distance of 3 to 5 mm. from the edge of the wound. The forceps may now be removed from the convex border of the tarsus and the lid margin be grasped at its middle point, a horn



FIG. 7—First act in the operation for removal of the tarsus without sacrifice of a fairly healthy conjunctiva (Kuhnt).

spatula being passed behind the everted lid, as shown in the diagram. A second incision, running the whole length of and parallel to the lid edge, is now made as nearly as possible in the healthy conjunctiva. Sometimes this will be three, sometimes even five mm. from the palpebral border, the intention

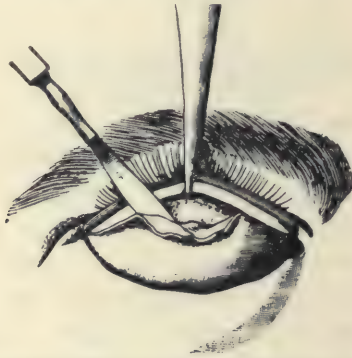


FIG. 8—Second act in the subconjunctival excision of the tarsus (Kuhnt).

being to remove as little of the unaffected mucous membrane as possible and so to leave as large a portion of the central conjunctiva area as is consistent with the needs of the case. The spatula may now be removed, the assistant drawing the lid upward and backward with one or two fixation forceps.

The operator then seizes the tissues at the nasal junction of the two incisions and with scalpel and scissors slowly excises conjunctiva and tarsus, carefully avoiding the orbicularis and Mueller's muscle. At this point the anesthetic may be removed, and time allowed for the bleeding to cease. I have not been much troubled with hemorrhage, although some small branches of the arterial supply may have to be twisted.

The conjunctival sac should be thoroughly irrigated and the lips of the wound brought together. To secure a satisfactory result one must be particular to place each suture in both wound margins so that it will be exactly oppose its fellow when the eye is closed. It is also requisite that the bulbar conjunctiva should not be too much put upon the stretch. The middle suture should first of all be tied with a single knot and it is wise to make certain, by closing the lid, to ascertain whether the precaution just mentioned has been taken before the final knot is tied. I would advise the operator to allow the patient to recover from the anesthetic sufficiently to enable him to determine accurately whether the palpebral movements are sufficient and to be sure that there is no irregularity visible at the lid margins. If the interpalpebral space is the same, both with the eye open and shut as it is on the opposite side, and if the lid margins have a regular outline, all is well and the threads may be cut off close to the knots.

As a rule there is little subsequent pain, and very little reaction. The *after treatment* is simple and need not interfere with the attention properly demanded by the presence of corneal ulcer or other complications. On the whole I find gentle irrigation of the sac four or five times daily with warm boric acid solution, followed by the instillation of warmed and steril vaseline, is grateful to the patient and acts very nicely. The vaseline keeps the sutures soft and serves to protect the cornea. I apply a light bandage over both eyes and order the patient to keep quiet, but do not insist upon his remaining in bed. The sutures are removed on the fourth or fifth day. In a week or ten days the wound is usually quite healed but the sac should be subsequently examined for the presence of granulation tissue or irregular wound margins. These are best clipped off or trimmed with scissors.

It is advised by some operators that no stitches be used,

owing to the possibility of corneal lesions. For my own part I have not witnessed these untoward results when the precautions I have mentioned have been taken, and I am sure that healing occurs sooner and the cosmetic effect is better when sutures are employed.

I do not see a great many cases of chronic trachoma, either in my private practice or in my dispensary work, and I have operated on only eight additional cases since my last report, but these have been so satisfactory to me that I take the liberty of reporting several of them here, choosing those instances that seem to me to illustrate the majority of the conditions in which, in my opinion, the operation should be done.

The most important result of this operation is the relief given to the irritative symptoms of the disease. Very shortly after the removal of the stitches we notice the subsidence of the photophobia, the lachrymation, the foreign body sensations and the local discomfort that accompany chronic granular lids, even when there is no ulceration of the cornea or no acute conjunctivitis present. Pannus is always lessened and may even disappear, and as a direct consequence of this the sight is much improved. In one very severe case of corneal infiltration, referred to in my previous communication, where the visual acuity had fallen to $\frac{1}{10}$ it rose to $\frac{2}{7}$ within three months after the tarsal excision. The asthenopia generally exhibited in the better eye of a case of chronic unilateral trachoma is wonderfully improved, and, strange to say, in not a few cases those common sequels of chronic trachoma, entropion and trichiasis, are decidedly less marked than before the operation. Finally, when we have to deal with recurrent ulcer of the cornea, a cure of the abnormal conditions behind it generally prevents a return of the disease.

The objections that have so far been urged to the removal of the tarsus are:

(1) That *ptosis* is likely to follow the operation owing to section or exsection of the levator palpebralis superioris. I have never seen a single instance of this sequel in the twenty-two cases in which I have myself operated on nor in patients under the care of others. Owing to the relief from spasm of the orbicularis palpebrarum (always more or less present

in all forms of chronic trachoma) the patient invariably opens his eyes to a greater extent than before, and I have not witnessed and do not expect, as the result of the removal of the tarsus, any diminution in the size of the interpalpebral fissure or in the power of the levator muscle. I believe the reason is that the attachment of the muscle is not to the tarsus alone.

(2) The same denial I would like to enter as to the probability of *entropion*. The fact that a shrinking and diseased tarsal cartilage is one factor in the production of lid edge incurvation accounts for the fact, sufficiently noticed by Kuhnt and seen in several of my own cases, that after the operation the previously incurving cilia gave no further trouble. In one instance, referred to in my first paper on the subject, and in another more recent one where epilation had been practiced for long periods, the eyelashes no longer required to be removed.

(3) *Ulcer of the cornea* has been noted after this operation, and it seems reasonable that the rubbing of the stitches and knots over the eyeball might produce an abrasion followed by infection. I have never had such an experience and do not think that if the directions for the operation, especially with the use of specially prepared sutures are followed, it is not likely to happen. Moreover, when one remembers that serious corneal ulcer may occur at any moment in these very cases, with or without treatment, it seems hardly proper to lay this accident at the door of the operation.

(4) The one complication to be avoided in Heistrath's procedure is the production of irregular, symblepharon-like folds in the region of the sulci. Unlike the alleged dangers just discussed, which are more imaginary than real, this is an accident which *may* happen to the careless operator. When it does occur it is very annoying to the patient, inasmuch as the ocular excursions are attended by dragging, drawing sensations, and in nervous subjects are exceedingly irritating. For relief of them it is best to dissect back the symblepharal attachments and implant mucous membrane or skin grafts to cover the denuded surface.

The following cases may serve to further illustrate the contentions of this paper:

CASE 1.—Sister V.'s eyes were infected from a patient in

a St. Louis hospital in 1893. She was treated during the following year by Dr. Alt and when I saw her, two years after, the disease had assumed the usual chronic form of the follicular type; the granulations were not exuberant, but corneal complications were frequent. The patient was unable to use her eyes and suffered much from foreign body sensations, headache and photophobia. She had numerous small *nebulæ* *corneæ* and a superior pannus on both sides. She came to me chiefly on account of pin-point ulcerations on the left cornea. I treated her with varying success by all sorts of methods and remedies for nearly a year. She had many relapses in the meantime and her visual power slowly declined. Finally, in December, 1897, I persuaded her to have both tarsi removed from the upper lid. This was done and the patient slowly recovered. To-day she has, with correction of her myopic astigmatism, $\frac{2}{7}$ vision in the left eye and a little better in the right eye. She reads words of Jaeger 1 with the right eye and of Jaeger 4 in the left. More important, her eyes are quite comfortable since the spring of 1898, when she had one corneal ulcer due, probably, to a number of infected follicles, which were not removed at the time of the operation and which I treated subsequently with *lapis divinus*.

CASE 2.—Mr. A. N. was attacked by inflammation of the lids in 1882 and until comparatively recently has been under treatment in France and in this country. I saw him in May, 1897, and found him suffering with well marked chronic trachoma in both lids, pannus in both eyes, a beginning staphyloma left (for which Galezowski had done an iridectomy), there were granulation masses in the left upper and lower folds, upon which I used the forceps. Then began a long, weary attempt to get rid of the disease. The patient was treated all summer. When I saw him in September, 1897, the secretion and granules were less, but he had had several pin-point ulcers in both eyes and was much discouraged. I did a Heisrath's operation on the left or better eye ($V = \frac{7}{200}$) and later on the R. The right lens becoming cataractous and the corneal staphyloma increasing, I removed the lens, with improvement. He began to get better and to-day is practically cured of his trachoma, but bears the scars of the contest. In left eye V with correction of $A = \frac{20}{70}$ —and words of Jaeger 4 at 10 cm.

CASE 3.—Oct. 21. Celia H., school teacher, had to relinquish her occupation on account of a chronic eye inflammation which affected both eyes in 1897. In September, 1900, I saw her, when she had a well developed follicular trachoma, affecting both lids of both eyes. V. R.=fingers at 1 meter: V.L.= $\frac{20}{30}$ —. There was total pannus R., by far the worse eye. Treatment was followed by improvement left but very little R. Heisrath's operation done R: many granulations found in the wound, but all lid movements good. Treatment with lapis after use of forceps in the left affected a cure. Patient returned a few weeks ago to say she had resumed school six months after the operation perfectly well. With correction V.L.= $\frac{20}{20}$. V.R.= $\frac{20}{70}$. There are nebulæ plainly to be seen on R. cornea but the operation scar is insignificant and there are no symblepharal folds.

CASE 4.—Maggie Driver, age 27, Versailles, Mo. Has had trachoma in both eyes for 12 years, with repeated exacerbations, corneal ulcers, pannus, etc. Father, mother and 11 brothers and sisters had disease. First saw her Nov. 7, 1902, when she had an acute attack of trachoma beginning four weeks previous. A well marked pannus present with some staining of left cornea with fluorescein. Right eye not so bad. Trichiasis both eyes. Right lower lid has been operated upon for entropion. Marked improvement under 20 per cent. argyrol used three times a week in office and a 5 per cent. sol. three times a day at home. Cold applications every 2 hours followed by boric acid lotion.

Jan. 7-8, 1903. Tarsus of R. eye removed.

Jan. 7-15. Tarsus L. eye removed.

Made an uneventful recovery.

LV. $\frac{5}{200}$. RV. $\frac{11}{200}$.

The excision of a part or the whole of the so-called tarsal cartilage, without sacrificing the overlying conjunctiva, is an operation suggested by Kuhnt in those cases where the disease has practically died out in the mucous membrane but is active in the submucosa and tarsus. I have had no experience of the method, although the *technique* presents no difficulties and the operation seems rational.

DISCUSSION.

DR. OSCAR DODD, Chicago.—What effect has this on the cul-de-sac? Perhaps I did not hear the paper as distinctly as I should, but I did not get the exact explanation as to that part.

DR. ALBERT E. BULSON, JR., Fort Wayne, Indiana.—I have been much pleased with this paper for the reason that it advocates an operation which will certainly give relief to a large number of cases suffering from the ill-effects of a thickened and inverted eyelid following trachomatous inflammation.

I wish to digress a little by reporting a case operated in a similar way to that described by the essayist. After repeated failures to secure satisfactory results in an aggravated case of entropion (the Hotz operation and several other measures being resorted to), I conceived the idea of removing a portion of the thickened cartilage which I thought in a very large measure responsible for the continued irritation of the eyeballs and practical loss of vision in both eyes from pannus. The operation was decided upon as being a measure that would produce no harm if it did not produce relief, for there was everything to gain and but little to lose in the treatment of the case. Under a general anaesthetic the conjunctiva was dissected up, and practically the entire tarsal cartilage removed, only a small part of the upper portion being allowed to remain on the theory that it might act somewhat as a support for the remaining tissues. The case went on to recovery, and I had the satisfaction of seeing the pannus clear up to a certain extent and vision improved so that the patient could see to get about without assistance, whereas before, his vision was so poor that but little more than shadows were seen. Some ptosis remained, but this was not sufficient to prevent vision, and the patient was so thoroughly satisfied with the results that he made no complaint regarding the slight drooping of the lid.

I am convinced that the operation is beneficial in a certain class of cases, but think it should be reserved for those obstinate conditions in which inversion of the lids continues in spite of all other treatment usually employed.

DR. ADOLF ALT, St. Louis, Mo.—I would like to ask what effect the removal of the tarsus has on the remaining granulating tissue. This operation leaves a strip of conjunctiva along

the upper edge of the tarsus and there is undoubtedly some trachoma in the tarsal fold, although there was not in the pictures. Is there no further growth of the trachoma? I have never seen a case yet in which I felt obliged to resort to this operation. It seems to me Dr. Prince read a paper on the same subject at our meeting some five or six years ago in Chicago. If it is of so much benefit we will have to adopt it as a legitimate measure.

J. B. WORRELL, M. D., Terre Haute.—I never had occasion to make the operation. I remember some years ago having seen a case in which the tarsus had been excised but do not know the technique in the case. Last year I saw a very unfortunate case in which the operation had been done and the patient had no lid left. Whether it was for the removal of trachomatous tissue or not, I cannot say. The problem was to restore the lid, and to a certain extent it was accomplished. I rise to ask two or three questions; in the operation, do you remove all the tissue between the two incisions, afterwards stitching the two edges together? (DR. WOOD.—Yes, where possible.) What is the effect of this upon the cul-de-sac? Would it reduce that to such an extent that it would limit the movements of the eyeball? Do you remove the tarsus entirely, leaving only the muscular tissue of the lid? (DR. WOOD.—Every bit of it.)

DR. CASEY WOOD (closing discussion).—The main question in cases of inveterate trachoma is, shall we go on treating them by the usual ineffective method, or try to eradicate the foci of the disease itself? So far as the formation of tears is concerned, the removal of the tarsus has not made any difference at all. In a few cases there was a complaint of "dry eye;" and one may easily believe that in removing a large amount of conjunctiva a considerable secreting area disappears; but it must be remembered that xerosis is frequently present in old cicatricial trachoma so that we may usually refer these dry sensations to the ravages of the disease. Except in one case, the author has never noticed any limitation of the upward or downward excursions of the eyeball. In simple excision of the folds of transmission for this disease, an operation often done and described by French authors and those of the German school, no mention whatever is made of subsequent limitation of the globular movements. Would it be an insuperable objection to the operation, even if it

did limit these rotations? External examination rarely shows that an operation has been done. In the majority of instances it is only when one attempts to evert the lids or to examine the condition of the folds that the absence of the tarsus can be noted at all.

The effect of tarsal excision on the remaining conjunctival tissue is very slight. However, the trachomatous tissue that remains can always be reached by remedies and I have not found any difficulty in so treating it, because it is superficial and can be reached by ordinary methods. But the nodules that are imbedded in or attached to the tarsus cannot be reached by mild means, and that is the reason for the operation. Our interest is to get rid of these active foci of the disease, which lie deep in the tarsus itself.

PARALYSIS AND PARESIS OF THE MUSCLE OF ACCOMMODATION.

By GEORGE F. SUKER, M.D.,

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IT is the endeavor in this paper to consider the paresis and paralysis of the muscle of accommodation as distinct and separate pathologic entities; and, to lay before the members sufficient valuable associated data so that our attention might more often be attracted to this particular lesion than has hitherto been our custom. Furthermore, it is intended to confine the consideration mainly to the so-called peripheral type. Topographical anatomy and physiological experiments warrant us to assume that the function of accommodation is an independent one with reference to the other functionings of the eye as such (Hess, 1902; Mauthner, 1899; Hensen and Voelckers, 1878). This does not make it necessary to exclude the so-called con-comitant associated acts of the iris and extraocular muscles.

The first correct conception or description of either paralysis or paresis, was given by Wells¹ (1811). He also knew full well the action of mydriatics on the power of accommo-

¹Wells, P. *Philos. Trans.*, cl., p. 378, London, 1811. (See Donders *Accom. and Refraction*, foot note p. 597).

dation. He did not, however, know of the various nerve centres concerned in the function of accommodation or pupillary reactions. This remained to be deciphered by Helmholtz³ (1853), Bruecke² (1846), Huebner (1872), Mannhardt⁴ (1858), Hensen⁵ and Voelckers (1868).

Prior to these days, paresis and paralysis of accommodation were often confounded with rapidly oncoming presbyopia (Himly⁶). Before the days of cerebral localizations and the physiology of presbyopia, it was not at all surprising to make this error, as in both conditions the near point recedes, and the far point remains about the same as before. Not only was it confounded with presbyopia but with many of the present day amblyopias and amauroses (Nagel, 1866, and Donders, 1866, mention this fact in a foot note). A dilated pupil is frequently a pertinent objective symptom in paresis and paralysis and at the same time accompanies many of the amauroses. It was, therefore, not at all strange for the older writers to often confound the two conditions. Especially as we now know paresis and paralysis of the muscle of accommodation is not such a clinical rarity.

From Himly's time (1811) down to Donders (1864) very little was said about these conditions of the ciliary muscle. In 1860, Donders⁷ gave the first accurate description of this ciliary affection as a sequel of diphtheria faucium (from an epidemic then in Paris). Since then, it has received more or less attention, but it was always associated with diphtheria and what is now called ophthalmoplegia interna.

Not knowing that the centre of accommodation is one in-

²Bruecke, E. Ueber den Musculus Cramptonianus u. d. Spannmuskel der Chorioidea; Mueller's Archiv., 1846.

³Helmholtz, H. Ueber d. Accommodation d. Auges; Archiv. f. Ophthalm. (Graefe), 1855. 1. 2.

⁴Mannhardt. Bemerkungen ueber d. Accommodationsmuskel u. ueber die Accommodation; Arch. f. Ophthalm., iv, 1858.

⁵Hensen and Voelckers. Experimentaluntersuchung u. d. Mechanismus der Accommodation; Kiel, 1868.

⁶Paresis and par. of accom. at this period was called "ciliary amaurosis" (Walther, Jour. von Graefe and Walther, Bd. 111, 22; "amblyopie presbytique" (Siehel Annal. d'Oculist. 1853).

⁷Donders. Paralytische Sympt. nach Diphtheria faucium; Archiv. f. d. holland. Beitrage z. Nat. u. Heilk., 1860, Bd. 11, 453. (note) Faure in the L'Union Med. 1857, mentions diph. par. in six cases and says that Trousseau, Bretonneau and Blache also observed it. Prior to this time, it was called "amblyopia" (Guimer, 1838; Ozanam, 1835).

dependent of the other eye centres, they never looked for paralysis or paresis as occurring unassociated with other intra- or extra-ocular paralysis. That they did not recognize this condition of the accommodation in ophthalmoplegia totalis, is evident from the writings of a certain Brunner⁸.

Though the muscular mechanism of accommodation had long been known (Brücke, 1846; Helmholtz, 1855), and some of its errors recognized, yet they failed to locate the lesions and centres. Neither did they interpret the occurrence of the lesions correctly. The condition was not even fully appreciated as late as 1878, as is evidenced by Hutchinson⁹. In an article of Ophthalmoplegia Interna, he prematurely places the lesion in the ciliary ganglion. To-day, we know that this ganglion has very little to do with accommodation whatsoever as a separate centre. The researches of Hensen and Voelckers have shown beyond a cavil that Hutchinson's assumptions were erroneous. Hutchinson evidently was not aware of the experiments of Hensen and Voelckers¹⁰ (1878), Hulke and Schweigger¹¹ (1858).

At the present time, we accept the facts regarding the centers of the accommodation and pupillary reaction as laid down by Huebner, 1872; Voelckers and Hensen,¹² 1878; Kahler and Pick¹³, 1881. These have demonstrated a series

⁸Brunner, C. De paralyti musculorum oculi nonnulla (Berolini, 1850, p. 10. He first used the term Ophthalmoplegia Totalis, but he does not speak of any condition wherein accommodation alone was affected and the other muscles intact.

⁹Hutchinson. "Ophthalmoplegia Interna;" read before the Roy. Med. and Chir. Soc., 1878. H. was the first to clinically speak of the terms "ophth. interna and externa." Graefe in 1868 uses the same term in speaking of analogous conditions. Eulenburg, 1871, speaking of similar affections of the third nerve appropriately applies the term "progressive" (very fitting for nuclear paralyses).

¹⁰Hensen and Voelckers. Vide general references, No. 2.

¹¹Schweigger and Hulke in 1858 demonstrated ganglion cells in the front of the choroid and ciliary region and associated them with the action of the iris. Graefe-Saemisch Handb. d. gesamt. Augenheilk., vol. 1. 1858.

¹²Hensen and Voelckers' (v. gen. ref.) arrangement: 1, Accommodation. 2, Sphincter iridis. 3, Rectus internus. 4, Rectus superior. 5, Levator pal. 6, Rectus inf. 7, Oblique inf. 8, Trochlearis.

¹³Kahler and Pick's arrangement (Prager. Ztschr. für Heilk. Bd. 11, 4, pp 301): 1, Accommodation. 2, Iris. Medial: 3, Rect. sup.; 4, Rect. ext. Lateral: 5, Levator pal.; 6, Rect. sup.; 7, Oblique inf. 8, Trochlearis.

Experiments on cats, dogs and monkeys have verified this classification.

of centers in the nucleus of the third nerve, which are associated with different functions. These centers lie partly in the posterior part of the third ventricle and partly beneath the corpora quadrigemina, in the floor of the aqueduct of Sylvius. They lie directly behind one another. That portion of the nucleus of the third, in the posterior extremity of the floor of the third ventricle just in front of the opening of the aqueduct, causes ciliary muscle contraction; a little farther back in the aqueduct is the iris contraction centre; the centre for the external ocular muscles is the next. From this arrangement, it can be said that in the loss of their functioning, we have:

(a). Loss of the power of accommodation.

(b). Loss of the pupil contractions.

(c). Paralysis of the muscles supplied by the third nerve.

Often the fourth and sixth are involved because their centres are contiguous to the third. However, an association of "a" and "b" constitutes an ophthalmoplegia interna. This ophthalmoplegia can be complete or incomplete, especially if we incline to Mauthner's view of the nuclei.¹⁴

Furthermore, the blood supply (Huebner¹⁵, 1872; d'Astros and Alezias¹⁶, 1892) to these intrinsic centres, as laid down by

¹⁴Mauthner. Ich bin auf Grund d. klin. Beobachtungen d. Ansicht dass d. Nerven f. d. Augenmuskulatur jedes Auges saemmtlich ihre Kerne auf d. gleichnamigen Seite haben u. d. daher d. einseitige totale Ophthalmoplegie einfach durch d. reihenweise Erkrankung d. gleichseitigen Nervenkerne bedingt wird. Die Lehre von den Augenmuskellähmungen, 1889, S. 368.

¹⁵Huebner. Zur Topographie der Ernährungsgebiete der einzelnen Hirnarterien; Centrbl. f. d. med. Wissensch. 52, S. 818, 1872. H. clearly shows that the circulation of the brain cortex and the base are entirely different. In the former innumerable anastomoses occur with an extensive ramification; in the latter, the vessels after a short course dip into the substance of the brain and supply the immediate surrounding areas without anastomosing. In other words, we have typical end arteries in the basal circulation and not in the cortical. They are end arteries according to Cohnheim. Each supplies a distinct, though small brain area; and, the supply of the third nucleus is no exception. The vessel supplying the nuclei of the iris and the accommodation, is the ramus communicans posterior and does not supply any other part of the third nucleus.

¹⁶d'Astros and Alezias dispute Huebner and say the blood supply comes from the posterior cerebral of the basilar and not directly from the basilar. It is called the arteria nucleoli oculomotorii. (A Les artères nourricières des noyaux du motaire oculaire commun et du pathétique; Soc. de Biol., Juin 1892).

these experimenters, will add material weight that one of these centres for the various functions of the third nerve can be affected without implicating the other two, in any shape, form, or manner. You can have a paralysis or a paresis of the extra-ocular or intra-ocular muscles, separately or combined; or, only one muscle in either group. But, it may be asked why it is that the iris and ciliary centres are more often involved than the others. No satisfactory reason can be assigned for this.

According to the foregoing, Hutchinson's theory that the ciliary ganglion is the nucleus for ophthalmoplegia, is evidently untenable. The ganglion would have to be divided in a manner like the third nucleus and this we know is not so. Experiments uphold Hensen and Voelckers but not Hutchinson. Again, it is very reasonable to suppose that a ganglion of the sympathetic group and not of the cerebrospinal, in addition to being removed from the nerve centres, has more to do with "intimate association" of closely allied automatic functions than with their differentiation from one another (W. A. Sturge, V. vf). Spec. ref. No. 8.

It may be stated as a fact (experiments in the past and the present bear out the assertion) that upon the removal of the ciliary ganglion¹⁷ and the stimulation of the cervical sympathetic¹⁸, the pupil still dilates to a considerable extent. The reverse holds true also. Furthermore, the pupil dilates when both mentioned ganglia are excised, and atropine is instilled or a peripheral irritation made. When eserine is instilled under these conditions, the catoptric images change. These points show that the iris must be supplied with dilating fibres derived from a different source than from the ones already noted. These accessory fibres are furnished by the ramus ophthalmicus of the fifth nerve. They enter the eye along with the optic nerve, upon the superior portion and external to the optic sheath. Division of the ramus causes greater pupillary reaction, i. e., contraction, than upon sectioning the ciliary or cervical ganglia. Hence, the iris must receive dilating fibres independent of the ciliary or cervical ganglia.

As the iris under these various conditions still responds to

¹⁷Adamück. *Centralbl. f. d. med. Wissensch.*, No. 28, 1876. Hensen and Voelckers, *Loco citato*.

¹⁸Hulke. See gen. ref.

eserine and atropine, and as these latter only affect nerve terminal centres, we are compelled to at least think of the ganglionic centres in the choroid, iris, and ciliary body as announced long ago by Hulke¹⁹, and Schweigger²⁰ (1858), and Meyer²¹ (1893). These plexuses possess the autonomy of nerve centres and hence will assist us in explaining many pupillary phenomena otherwise inexplicable.

Accepting these facts regarding the peripheral and central nerve centres for the accommodation and iris reaction, it remains to be demonstrated whether the sympathetic nerve has any connection with the accommodation. It is an axiomatic fact that the motor oculi is the nerve of active accommodation and ciliary muscle contraction. Also an axiomatic fact, that a paralysis of a part of the third nerve causes a paresis or paralysis of the act of accommodation. In vain have experimenters tried to prove the sympathetic nerve supply for it. The sympathetic fibres were supposed to be inhibitory accommodation fibres²². It would be truly convenient to have this inhibitory supply in explaining many apparently unconnected symptoms regarding lesions in these various centres.

Morat and Doyon, upon cutting the sympathicus and stimulating the cut end, noticed an enlargement of the catoptric images and concluded therefrom that the nerve acted as an inhibitor and assisted in the accommodation for distance. Upon repetition of this experiment by others²³, it was found not to prove true. Langley and Anderson emphatically deny Morat and Doyon, and are upheld by Hess²⁴, Rømer and Dufour²⁵.

¹⁹Schweigger. *Loco citato*.

²⁰The writer has many times noticed this condition while experimenting with these ganglia in reference to glaucoma.

²¹Meyer. *Zur Kenntniss zum Bau der Iris*; *Biolog. Untersuch. Neue Folge*. V., 1893.

²²Morat and Doyon. *Le grand sympathétique nerf de l'accommodation pour la vision des objets éloignés*; *Compt. Rend. de l'Acad. des Sc. and Archiv. de Physiol.*, III, 507, 1891.

²³Langley and Anderson. *On the mechanism and movement of the iris*; *Jour. of Physiology*, XIII, 6, 1892.

²⁴Hess. *Arbeiten aus d. Gebiete d. Accommodationslehre*; *Graefe's Archiv. f. Ophth.* XLIX, 2, 1899. *Graefe's Archiv. f. Ophth.* XLII, 1896, and, XLIII, 1897.

Hess. *Bemerkungen zur Accommodationslehre*; *Centbl. f. prak. Augenheilk.*, July, 1899.

²⁵(Dufour and Rømer assisted him (Hess) in this work).

These fibres were supposed to call forth a decided flattening of the lens through their action on the inner layer of the ciliary muscle. Though there are two distinct muscle layers, an outer and an inner, they act as a single muscle²⁶ (Bruecke in Mueller's *Archiv.*, 1846, p. 370). As far as we know their contraction causes a drawing forward of the ciliary body and processes, a relaxation of the zonula of Zinn, a swelling of the anterior surface of the lens and a sinking of the same. Hensen and Voelckers needle experiment²⁷ absolutely proves that there is no muscle in the eye purposely to assist in focusing it for the distance. The catoptric images also seem to disprove this assumption.

Having determined that the ciliary muscle, the third nerve, and a separate central nucleus controls the accommodation, we may ask what relation the action of the iris sustains to this function. The contraction of the pupil during accommodation is the oldest known symptom in connection with it (note).

It was long thought that accommodation depended on the contraction of the iris. However, Koster²⁸, and before him Weber and Hess²⁹ had determined the pupil contraction to be

²⁶That the ciliary body was muscular, was a well known fact to Kepler in 1611; Eustachius in 1722, even had a diagram thereof. Porterfield (1759), Morgagni, Briggs and others knew of the action of the ciliary muscle. Zinn, however, denied that they ever knew the existence of the muscle. In 1846, Bruecke described the "tensor choroideae;" about this time it was also described by Bowman. In 1856, H. Mueller described the inner muscular layer of the ciliary body.

²⁷H. and V's., experiment. They inserted needles into the equator of an enucleated eye (and earlier into the living animal) then electrically stimulated the ciliary body region and watched their motion as follows: One needle advanced forward, showing advancement of the choroid; the one through the ciliary did not move; one very near the macula appeared to remain perfectly quiet (latest references to this experiment is made by Hess, in Graefe-Saemisch, 1902)

NOTE.—Scheiner (1619), Morton and Haller (1769), tried to explain all accurate accommodation by the action of the iris. It was Hering and Donders who demonstrated the synchronous action of pupillary and ciliary contraction during accommodation, but they did not say whether the pupillary contraction was a reflex act or not during accommodation, which it truly is.

²⁸Koster. *Bemerkungen zu den Versuchen von Hess ueber d. Accommodation Archiv. f. Ophthal.* XLVII, 1., 1898.

Koster. *Ibidem; Entoptische Beobachtungen; Archiv. f. Ophthal.* XLVI, 1., 1898.

²⁹Graefe-Saemisch *Handb. d. gesamt. Augenheilk.*, Bd. VIII, Kap. XII (1902).

only associated with convergence and not accommodation. Tscherning³⁰ regards the accommodation pupil contraction as a mechanical manifestation. At one time it was thought that the contraction of the iris was due to the amount of blood in it during the act. This idea is untenable, as it has never been demonstrated. The blood pressure in the iris and the ciliary body depend upon their inherent contraction activity as is seen in any other muscle. The contraction of the iris is not an essential factor in accommodation, but only an associated physical phenomenon.

Some facts have been set forth which warrant us to speak of paresis or paralysis of accommodation as a separate manifestation. From the foregoing we can look upon this affection of the accommodation at times as independent of internal or external ophthalmoplegia. It matters little whether you accept the Young³¹-Helmholtz or the Tscherning³² theory of accommodation, the symptoms are the same.

With a paralysis or a paresis of accommodation, it is then not so strange to have the other so-called associated functions involved to a certain degree. The loss of the function of any one of these three materially influences the extent of action of the remaining two. This difference in the functioning of the other two may, in many instances, be only apparent. (It is not deemed necessary to enter upon the actual mechanism of accommodation, only a brief outline³³ is needed).

³⁰He demonstrated a dilatation and a contraction of the pupil on the cadaver by inserting a hypodermic syringe and gently drawing the piston forward and backward.

³¹Young demonstrated accommodative changes to take place in the lens by experiments now renowned (On the Mechanism of the Eye, Phil. Trans. 1801).

³²Vide gen. ref. No. 6.

NOTE.—Descartes. 1637, was the first to associate convexity of the lens with accommodation (Trans. de homine). Mauthner gives credit for this to Scheiner in 1619 vide M. Augenmuskellaehnungs Lehre, 1889.

³³The act of accommodation ensues as follows: 1. Stimulus from central portion of third nerve nucleus. 2. Contraction of ciliary muscle. 3. Slight forward traction of chorioid and perhaps of retina. 4. Relaxation of the Zonula. 5. Increase in the ant. convexity of lens, with lessened vertical and increased antero-posterior axis. 6. Sinking of the lens. 7. Decrease in the depth of ant. chamber. 8. Trifle forward movement of iris. 9. Perhaps the vitreous also moves forward. The associated functions are: 1. Contraction of iris. 2. Convergence, with a trifle turning downward of the eye.

From a standpoint of practical symptomatology, there is very little difference between a paralysis and a paresis³⁴. Yet, pathologically often a great difference is manifested. A paresis is frequently a partial functional loss or inability, with no demonstrable lesion, excepting those perhaps peculiar to exhaustion³⁵. A paralysis on the other hand is very often the result of active pathologic changes—peripheral, central, or both.

We must differentiate between an active and a passive variety of paresis or paralysis of the act of accommodation. That is to say, active when the ciliary muscle or nerve function is involved; passive when the lens or the zonula alone are implicated. In the active, we distinguish a myopathic and a neuropathic type. Again, the active type is either a peripheral, a central nucleus, or an orbital lesion. The paresis is more often a peripheral lesion (myopathic) rather than a nuclear or orbital; while the paralysis is apt to be a nuclear or a peripheral nerve lesion.

The changes in the passive portion yield the same symptoms as those in the active part. Therefore the two conditions are easily confounded³⁶. Any change in the passive part, either in the lens or zonula, is demonstrable by the shrinking of the total range of accommodation. However, not every ciliary muscle paralysis is evidenced by a receding near point and a shortening of the accommodation range.

Either the active or the passive variety may appear as a separate manifestation, unassociated with any other symptom. But as a symptom itself, only the active variety can be associated with an ophthalmoplegia interna, externa, or

³⁴An exhaustion of a muscle is often similar to a paresis. At times they are not to be differentiated.

³⁵Paralysis. The suspension or abolition of functional power, especially in the nervous system, in which case there is a temporary or permanent loss of the power of motion or sensation or both, in the parts supplied by the affected nerve.

Paresis. An incomplete paralysis, especially when not associated with any demonstrable organic lesion—limited to motion and not to sensation (Foster).

³⁶Accommodation paresis is not the same as ciliary paresis. This is evident in presbyopia, which is an accommodation paresis but not a ciliary paresis. A normal eye can have the normal range of ciliary contraction, yet the altered lens will not respond.

totalis. In the former two it may be absent, but in the totalis it must be present.

To make the diagnosis of paresis or paralysis of accommodation, we have to take into consideration the actual range of accommodation and to determine the range of the accommodation which is characteristic of that age of the patient in whom the paresis occurs. This latter is dependent on the passive part of the accommodation. On account of the increased hardness in the lens with age, so in proportion does the contraction of the ciliary muscle become optically less evident. In old age the effect of this contraction is entirely latent. Hence it follows that any ciliary muscle contraction is more readily recognized in the decreased accommodation range in the younger individuals, as they have very little that is latent. In the aged,³⁷ say 70, the ciliary muscle can be absolutely paralyzed and yet no change in the range of accommodation or recession of the near point takes place.

The objective proof of a ciliary muscle paresis or paralysis is only demonstrable when it is greater than the range of latent accommodation. In other words, it must reach such a degree that by the utmost contraction of the ciliary muscle a complete relaxation of the zonula is not obtained. Again, any decrease in the manifest accommodation (peculiar to the age of the patient at the time) is evidence of the presence of a ciliary paresis or paralysis. But on the contrary, not every ciliary paresis or paralysis is evidenced by a corresponding decrease in the manifest range of accommodation. This point is well demonstrated in presbyopia.

It is also worth noting that a graduated scale for the amounts of paresis or paralysis can not be established as for presbyopia. Though, in a sense presbyopia is a paresis of the ciliary muscle according to Foster³⁸ and Kirk³⁹. One rea-

³⁷Presbyopia is a physiological lens hardening and never (so many say) due to any changes in the active portion of the accommodative mechanism. It is an open question whether an abnormal early hardening of the lens ever takes place, which can simulate presbyopia. It must be a hardness not due to any disease or complication.

³⁸Foster, M. Text-book on Physiology, 1891, 6th ed., p. 47, says: "In presbyopia the failure or loss of accommodation may be due to a loss of the elasticity of the lens, or an increasing weakness in the ciliary muscle or to the parts becoming rigid."

³⁹Kirk holds the identical view as Foster (Hand-book of Physiology, p. 71).

son is that the gradual recession of the near point and the latent range of accommodation to become manifest is a physiological process, progressing uniformly;⁴⁰ the other condition is a variable pathologic process, decidedly unequal.

In young people the amount of paresis can be approximately measured, but beyond 65 our present methods fail to establish whether the ciliary muscle is paretic or paralytic. Therefore ciliary paralysis is only of diagnostic import or becomes recognizable as a distinct manifestation in practically the "presbyopic." Then, too, only when the implication is greater than the latent range of accommodation does it annoy the patient.

As already said, a paralysis or paresis of the accommodation or of the ciliary muscle may yield the same clinical picture. Therefore, we must be careful in using this symptom as a diagnostic or prognostic point.

Mydriasis, complete or partial, frequently accompanies a paresis or paralysis of the ciliary muscle. Yet, as we have shown, one action is independent of the other. In a few instances a myosis was noted, with a paretic ciliary muscle⁴¹.

In the peripheral paresis or paralysis, the ciliary body or processes are directly or indirectly involved⁴². The neuropathic involvements are frequently central, or peripheral while the myopathic are peripheral.

In general, the symptoms of a paralysis of the muscle of accommodation is dimetrically opposite to that of a spasm. The extent of the involvement depends upon whether the eye is emmetropic, brachymetropic, or hypermetropic; and, whether the patient is presbyopic or not. Paresis on the other hand simulates more closely what we term asthenopia, and often unable to be differentiated with our present day methods. The disturbances are greatest in the hypermetropic, as both near and far points are affected. Next comes the em-

⁴⁰One 25 years old with a normal near point can have as much ciliary paresis as a child of 10 or 15 whose manifest accommodation range is about one-half the normal and whose near point is correspondingly removed.

⁴¹This condition must be one form in which there is a central and a peripheral lesion.

⁴²There may be a complete rupture of the zonula giving the same symptoms as a complete paralysis, yet the ciliary function is intact. Even vitreous disease has been reported to cause a relaxation of the zonula and resemble a paresis (Mauthner, Donders).

metrope, in him the near point is perhaps only farther removed than normal. The brachymetrope suffers the least, especially if the amount of error be 3 or 4 dioptries and no glasses were ever worn. In him, complete paralysis may take place and no subjective symptoms be complained of⁴³.

The annoyance is not very marked if the lesion is limited to one eye. A frequent accompaniment is micropsia. As already stated, other symptoms are often associated with this condition of the ciliary muscle, that we can provisionally tabulate them as follows:

1. Accommodation alone affected—incomplete ophthalmoplegia⁴⁴ interna.

2. Accommodation and iris affected—complete ophthalmoplegia interna.

3. Accommodation, iris and external eye muscles—ophthalmoplegia totalis.

Any of these conditions may be a nuclear, a peripheral or an orbital lesion. The nuclear type of paralyzes, especially of the progressive order, often have as their first symptom, a paralysis of the accommodation⁴⁵ (Mauthner, Graefe, and others). These nuclear affections may be bilateral or unilateral, complete or incomplete (vide foot note on ophthalmoplegia). The orbital lesions are more readily understood than the nuclear, as we may look upon them as peripheral in nature.

It is indeed difficult to explain how some constitutional or even brain lesion will practically isolate and only implicate the nuclear centre for accommodation. It can only be explained upon the basis of Huebner, Hensen and Voelckers. The nuclear paralysis or paresis may have the following brain lesions as its cause: hemorrhage, embolism, meningitis,

⁴³This is particularly noticeable should a small amount of ciliary power be left. On account of the effort of the residual power of accommodation, the objects seem nearer and therefore smaller.

⁴⁴The term ophthalmoplegia is, generic, having reference to the paralysis of any of the ocular muscles, intra- or extra-ocular. Mauthner (*Die Lehre der Muskellähmungen*, p. 306-307) gives this very comprehensive classification:

Central or Peripheral	} ophthalmoplegia	{ perfecta imperfecta	{ unilateralis bilateralis ibidem	{ exterior interior ibidem

⁴⁵The case of Heinrich Heine, the German Poet, is a celebrated case of progressive nuclear paralysis.

thrombosis, tumor, abscess, cerebral syphilis, internal hydrocephalus, and posterior spinal sclerosis. Syphilis is by far the most frequent underlying cause for the nuclear implications. However, not in all of these is the entire centre of accommodation involved, unless a complete ophthalmoplegia ensues.

The ciliary muscle itself may be incapable of reacting in the proper proportion to the nerve stimulus and this would evidence itself as a paresis rather than a paralysis. This may be due to inflammation and its results. Senile changes in the ciliary muscle can produce a paresis. The constant inactivity of the ciliary muscle in the various forms of strabismus can give rise to a paresis or even a paralysis. Excessive nutritive disturbances may also call forth a condition not unlike a paresis of the ciliary muscle.

The various conditions which may give rise to a paresis or a paralysis are as follows:

SYPHILIS.

Under this heading we may include tabes. The ciliary muscle is very often the first that suffers in tabes. The paresis, more often though a paralysis, is bilateral. Unilateral paralysis of the accommodation without mydriasis but with the characteristic anesthetic temple areas is also seen in tabes (Galezowski⁴⁶). In the majority of the Argyll-Robertson pupils⁴⁷ will a paresis of accommodation be found. In simple confirmed syphilis, the accommodation implication is very late in manifesting itself, if at all. Brain syphilis frequently involves the accommodation centre very early, often being periodic and recurrent. This is not very strange when we consider the blood supply of this centre and accept the view of Adamkiewicz⁴⁸. But why the centre for the intra- or extra-

⁴⁶He considers this condition almost pathognomonic of tabes.

⁴⁷The Argyll-Robertson pupil is a nuclear lesion, often attributable to a hemorrhage. From here the lesion travels from the aqueduct of Sylvius to the floor of the third ventricle and destroys the connection between the third and first nerves without affecting the sphincter of the iris.

⁴⁸Adamkiewicz has demonstrated that the sclerotic process in tabes is determined by the course of the arteries, especially in the brain. (The original is not accessible—quoted from Schmeichler, Knapp Archiv. Bd. XII. p. 335-364, 1883). The original article is: "Die Blutgefäesse des menschlichen Rückenmarkes; Sitzungsab. der Academie der Wisschf. in Wien, 1882, Bd. XXXIV.

ocular muscles are so frequently involved in the early stage of syphilis can not be explained, unless it is due to their peculiar blood supply. Graefe says frequent recurring accommodation paresis with mydriasis, and alternating bilaterally, should lead one to suspect mental disease, especially if syphilis is present.

Usually the paralysis is bilateral and accompanied by either a partial or complete mydriasis. This is particularly true in tabes (Galezowski). Exceptions to this, however, are noted by Alexander, Kirmisson, Jeaffreson, Hosch, Ferron and Mauthner (vide special ref.) Hutchinson in his early reports (*Med. Chir. Trans.*, 1878), mentions unequal involvement of the power accommodation in syphilitics. The specific involvement of accommodation is more apt to be central than peripheral and often depends on minute hemorrhages into the nucleus. This condition has been demonstrated post-mortem in a number of cases. All the sudden ophthalmoplegias with recurrences are indicative of syphilitic nuclear lesions, rather than peripheral.

Inherited syphilis does not as frequently cause a paralysis as the acquired form (*St. Bernheimer*, gen. ref. No. 21, p. 14). A latent form of syphilis can be recognized by a paralysis of accommodation with pupillary differences or bilateral mydriasis. These latter two conditions are often for a long time the only expression of a syphilitic infection. However, syphilis, especially of the nervous system, is more prone to involve the extra-ocular muscles than the intra-ocular, giving an ophthalmoplegia externa (complete or incomplete).

J. Hutchinson's writings show very clearly that the ciliary muscle implication is very rare in inherited syphilis.

The prognosis in this class of cases is not very favorable or even encouraging. Alexander (vide special ref.) does not believe a cure ever takes place.

SYMPATHETIC OPHTHALMIA.

In this direful disease ciliary muscle paralysis may be a very early manifestation in the sympathizing eye. Fuchs and Cuignet lay considerable stress upon its being fairly diagnostic, providing conditions for sympathetic ophthalmia are present. The writer can affirm this observation in a case

which he had under treatment. How this ciliary paralysis is brought about is a question. Randolph's researches on the influence of toxins in inflammations of the eye are of paramount interest in this connection. Whether it is a peripheral or a central paralysis has not been stated. The writer considers it a pure peripheral lesion, as are so many of the toxic type.

Perhaps it is a neuropathic paralysis. The mydriasis, which often is of a moderate degree, is likewise a peripheral lesion. In sympathetic irritation this ciliary complication has not as yet been reported, hence in doubtful cases this symptom will prove of some value.

LOCAL PERIPHERAL CAUSES.

The paresis or paralysis here are usually secondary to some disease in the iris, ciliary body, choroid, or retina. If it follows one of the mentioned causes it is of the myopathic type, and it is then the result of plastic exudates or organized inflammatory material binding down the ciliary body and processes. Atrophy of the muscle is then prone to follow these changes. It is not a want in the nerve stimulus, but an inability on part of the muscle to contract. This condition may account for the reduced vision in such cases that still have clear media and no other complications to explain the reduction.

Glaucoma frequently produces a paralysis or paresis of the accommodation. More often it is a paresis. We are all familiar with this symptom, and often have it as a premonitory sign, recognizable by the frequent desire to change the "glasses." This condition is more often witnessed in that form of glaucoma wherein we have a shallow anterior chamber and a markedly dilated pupil. However, it is quite evident that the passive part of the accommodative mechanism suffers to a certain extent as well.

Injuries in the immediate vicinity of the eye frequently call forth a paralysis of the accommodation. Injuries to the cranium may yield nuclear accommodative interferences, with more of a general ophthalmoplegia. Very often there is a complete rupture of the zonula, which will cause a passive paralysis, as the ciliary muscle is left intact. This form of

paralysis is not rare, as is evidenced by Power, Harlan⁴⁹ and others.

Defective teeth are also responsible for ciliary paresis. Schmidt-Rimpler and Schmidt (1868) have cited a series of cases of asthenopia of the paretic type that were due to defective upper teeth. This is a peripheral reflex paresis. Schmidt⁵⁰ is inclined to think it very common indeed. Few cases were observed by the writer. No recent reports are at hand. Linnell⁵¹ (1884) reports a case of paretic ciliary accommodation as being due to an irritation of the superior maxillary nerve. This must be the basis for the other cases as well.⁵²

Over-exertion of any description is a fruitful cause for paresis of the ciliary muscle. It is peripheral and perhaps many of the accommodative asthenopias come under this heading. Any strenuous occupation or the so-called misuse of the eyes may give rise to it. Panas⁵³ mentions a case of unilateral paralysis with amblyopia as the result of undue exposure to intense electric light. Working over bright fires seem to cause it also (Colsman).⁵⁴ Mydriasis as a rule does not accompany this form. Students, typewriters, and electricians form a large contingency.

This variety is mainly due to the accumulation of waste energy material. Hutchinson's⁵⁵ cases (v. special ref.) following shock and lactation will properly belong to this category.

Poisons, either local or general, very often produce paresis or paralysis. As to the local ones, we need only mention the cycloplegics and their allied group of remedies. It is often a very important symptom in meat, fish or vegetable poisonings. It is more in the nature of a marked paresis than an outright paralysis. Mydriasis is often an associated symptom. The agent is invariably a ptomain and acts peripherally, in a manner similar to the cycloplegics. Therefore it is apt to be

⁴⁹Harlan and Power. Vide special ref.

⁵⁰Schmidt. Vide special ref. He found 73 cases in a series of 92.

⁵¹Linnell. Vide special ref.

⁵²The disease travels by way of the spheno-palatine and ciliary ganglion, the connecting link being a small nerve fibre.

⁵³Panas. Vide ref.

⁵⁴Colsman. This is a very unique case. Vide ref. No. 9.

⁵⁵Schapringer's case of paresis with apparent myopia may be of this class, though he does not say so, yet the history points to it.

a nerve paralysis. Very often it is the first symptom which manifests itself in these cases. A like condition has been noted in tyrotoxicon poisoning (Fisher, Fuerst, Weiss, Leber, Knies, Groenouw, *vide special ref.*). In this particular we must not forget to mention the tobacco, alcohol, cocaine and morphine habits. In alcohol it is a paresis from the beginning, while in tobacco, it is a spasm first, which later is followed by the paralysis. In cocaine, we have a paresis followed by a complete paralysis.

Let us now consider some of the diseases such as diabetes, diphtheria and the like with reference to ciliary paralysis and paresis.

DIABETES⁵⁶.

Very frequently diabetes involves the accommodation apparatus⁵⁷. Whether this involvement is central, peripheral, or due to the accompanying reduced muscular vitality which is common to diabetics, is still a debated question. No doubt we can accept all three causes at times. It is usually a paresis and seldom associated with mydriasis. Von Graefe first called attention to this lesion in 1858 and since then many observers have recorded cases. Often it is among the first symptoms. Many regard it as a toxic manifestation and the writer shares the same view. For, upon the decrease in the glycosuria the intra-ocular paresis rapidly improves⁵⁸. Jacobson looks upon the paresis as a peripheral neuritis or as a hemorrhage. Foerster, as many others, regards it as due to the general muscular weakness. It may be a nuclear lesion and then unilateral (perhaps a hemorrhage). The perverted presbyopia must not only be looked upon as a ciliary paresis but also as due to changes in the lens itself.

INFLUENZA⁵⁹.

The interference of accommodation was among the first "so-called out of the ordinary symptoms" recorded. At times

⁵⁶Berger, E; B. first advanced the toxic theory in diabetes, not only of the intra, but also of the extra-ocular muscle affection. He assigns his reasons in a very valuable contribution (*vide ref.*).

⁵⁷Galezowski. In 1883, in a thesis, he says 7 per cent. of all diabetics have some muscle paralysis. No doubt some are nuclear hemorrhages.

⁵⁸Knies. K. favors the acute intoxication theory. (It does seem a rational view both here and in albuminuria). See note on Berger.

⁵⁹Williams. R. A. (*v. special ref.*) gives a detailed account of paralysis and paresis in influenza, with a review of facts. Statistics are very meager, but would not be, were the cases reported.

the paresis or paralysis, as in diphtheria, appears some time after the patient is entirely well. Very often though, it comes on during the progress of the attack. Rarely does it appear as a premonitory symptom. The disease being of germ origin, we can well attribute the accommodation interference of some toxin, just as in diphtheria. It apparently is of a peripheral type; when brain complications arise early in the disease, then nuclear implications are prone to occur.

The affection may be unilateral, but as a rule it is bilateral. The paresis occurs very often when the nervous manifestations are pronounced or marked muscular enfeeblement follows convalescence. Iris complications may be absent and usually are very slight if present at all. This paresis or paralysis may be a part of a more extensive ophthalmoplegia⁶⁰, either interna or externa. If so, then brain symptoms are invariably noted. The prognosis ordinarily is very favorable.

DIPHTHERIA⁶¹.

A very large quatum of accommodation interference is furnished by diphtheria. The severity of the diphtheria bears no relation to the frequency or severity of the paresis. Mild attacks often are followed by the most protracted forms of paralysis. The young and the middle-aged share alike. Frequently pharyngeal and laryngeal pareses accompany the other. It generally makes its appearance when full convalescence has been established. The onset is usually sudden; the attack continues from ten days to two or three weeks. Recovery is the invariable rule, and this without specific medication.

It is often a valuable diagnostic point in doubtful cases of diphtheria. The infection must have been diphtheritic, to the contrary notwithstanding, when it follows a sore throat. No angina⁶² produces a similar paresis or paralysis. It is not necessary to have the classical diphtheria in order to get this

⁶⁰Uhthoff. (Deut. Med. Wochschr. No. 10, 1890). U. cites a case of ophthalmoplegia interna. Gayet (Jahr. f. Aug. 1876) cites a case of total ophthalmoplegia externa, without implication of the levator palpebræ superioris. Guttman. Loco citato.

⁶¹The largest statistics on this point are furnished by Moll and Remak (v. special ref.).

⁶²Bass. Monat. f. Augheilk., 1886, p. 273. A case due to mumps (?) and sore throat.

complication. A diphtheritic wound anywhere can give rise to it.

Some very peculiar cases are recorded in this particular⁶³. If the accommodation interference is associated with absence of patellar reflex, tabes may be very closely simulated⁶⁴. The attack is usually bilateral, though unilateral⁶⁵ and ^{3a} cases are on record (often accompanied by other symptoms).

The introduction of antitoxine did not abate the frequency of post-diphtheritic paralysis as was hoped⁶⁶. The administration of it for the paralysis is as good as useless⁶⁷. It does not cut the attack short, nor does it transfer a paralysis into a paresis, as was thought at one time. Behring never took this complication into consideration when he was experimenting with the serum. There is no reason why he should have, as the prognosis is at all times absolutely favorable. But one fact is evident, namely since the serum, the post-diphtheritic paralyzes are more frequent. Statistics, however, seem divided on this score. Schmidt-Rimpler holds that the antitoxine hastens the return of the accommodation. This has not been the observation of others nor of the writer.

Rosenmeyer⁶⁸ and Boerger⁶⁹ in a large series of cases say that the serum has reduced the number of paralyzes. Perhaps if the serum were given in the very outset of the disease and not on the third or fourth day as is so common, the number of paralyzes might be less. The delay gives the toxins a chance to become disseminated and a severe attack is not needed for a production of these pareses. St. Bernheimer

⁶³Gayton, W. In the London Lancet describes a case of post-diphtheritic accommodation paralysis following genital diphtheria (v. special ref.)

⁶⁴Jessop, H. W. V. special ref. The only case on record.

⁶⁵Dufour. He cites a case of monocular diplopia following diphtheria. It must have been of the myopathic type and involving only a part of the muscle. (v. special ref.).

^{3a}. Wolfe. He says "both eyes are rarely affected" (1882). The statistics of Moll and Remak do not uphold him. (v. special ref.).

⁶⁶Janowski (v. ref.). "Die Kranken werden jetzt die Laehmungen wohl oeffters erleben als vor der Serumperiode und dementsprechend wird also die absolute Zahl derselben seit Einfuehrung letzterer steigen."

⁶⁷Hertel and Graefe (v. special ref.) both maintain this idea and cite cases in support of their view.

⁶⁸Rosenmeyer. Wien. med. Wochenschr., 1886, No. 13 and 14.

⁶⁹Boerger. Ueber 100 Faelle von Diphtherie mit Serum behandelt, Dent. med. Wochenschr., 1895, No. 25.

inclines to the view that the serum has a beneficial influence on the paralysis. The paresis may cause a persistent asthenopia, though it never remains permanent⁷⁰.

The diphtheritic paralysis is a peripheral one⁷¹. Hensen and Voelckers place the lesion in the nerve endings. Knies⁷² says it cannot be a hemorrhage or a terminal nerve inflammation but some toxin which directly affects the ciliary muscle. This belief is now accepted by quite a few clinicians⁷³.

HYSTERIA⁷⁴.

In hysteria a spasm of the accommodation is more often seen than a paralysis. Charcot and Parinaud have recorded unilateral diplopia as due to a paresis or a paralysis of the muscle of accommodation. Micropsia is very often complained of in this condition while it is rare in the other forms of paralysis. Here, too, the mydriasis is apt to be unequal. Children furnish the largest quota of cases, and in them it is the first symptom of a general hysterical manifestation. Perhaps spasm and paralysis are of like frequency, the former in the nervous and the latter in the plethoric type of hysteria. So long as the far and near point are not coincident, the patient will enjoy fair vision. This depends a great deal upon the static refraction of the eyes. Very few hysterical patients ever escape without accommodation interference. Parinaud holds that paralysis is rare in hysteria, though Galezowski and others have the opposite view. Both are right in a measure.

GENERAL DISEASES.

In the greater majority of cases it is a paresis rather than a paralysis. Many of the infectious diseases as typhoid, ma-

⁷⁰St. Bernheimer. *Aetiologie u. path. Anatomie d. Augenmuskellaehmungen*; Graefe-Saemisch Handb. d. gesam. Augenheild., bd. VIII, S. 16-80, 2te. Ausgabe.

⁷¹In the days of Donders (1860) and Nagel (1866), they did not know whether it was a peripheral or central lesion. For them unilateral paralysis confirmed peripheral, and bilateral paralysis, central lesions. This is obviously incorrect.

⁷²Trans. of Ophthal. Soc. U. of K., vol. vi, 386.

⁷³This view coincides with those of Randolph; Amer. Med. Sc., Nov., 1902.

⁷⁴Donders speaks of this condition as "painful accommodation."

(a). Foerster designates it as "hysterical kopiopia."

(b). Nagel calls it "hyperaesthetic ciliary muscle."

laria, pneumonia, scarlet fever, measles, whooping cough, neurasthenia (Grand-clement), scorbutus (Bristowe), and multiple neuritis (Oppenheim) develop a paresis either during the attack or directly upon convalescence. The cause here is the general muscular enfeeblement that usually accompanies these diseases. We can regard the most of these cases as a paresis due to exhaustion of the ciliary muscle, hence myopathic, and not as a motor oculi nerve interference, and therefore not a true paralysis. No doubt the toxins which circulate in the system as a result of the disease play no minor role in producing these pareses.

The implication of the ciliary muscle in this class of cases is of the milder variety, and a rather rapid recovery follows the toning up of the general muscular system. These patients complain of a severe accommodative asthenopia. In fact, it so closely resembles this, that many a pair of glasses is prescribed, which would not have been necessary had the patient had the proper care and attention immediately following the sickness. It is simply to avoid this ciliary paresis that we prohibit the patients from reading or using their eyes very much during any protracted illness.

Many of the anaemias (as chlorosis, simple anaemia, and leukaemia,) are prone to have some accommodation trouble and usually it is a paresis of the ciliary muscle; this is particularly true in chlorosis. Much suffering could be spared the chlorotic if the physician would but recognize this point. A proper correction will relieve the trouble, allow the ciliary muscle to recuperate; and, upon the cure of the chlorosis, the glasses are in most cases not needed.

Rheumatism, gout and dysentery lay claim to quite a few cases of ciliary paresis. Even the involvement of the accessory sinuses of the nose are responsible at times for a paresis of the ciliary muscle.

Strange, that tuberculosis with all its disseminating tendencies seldom causes a paresis, excepting that which may be due to muscular feebleness. There are many more diseases in which we may have this ciliary muscle implication, but the ones mentioned will suffice. The writer did not intend to dwell at all upon purely nuclear or orbital lesions, though reference has been made thereto.

In general it may be stated that a peripheral paresis and paralysis develops slowly as compared with the nuclear type. This serves as a point in differential diagnosis at times. Again, in the peripheral varieties mydriasis is often wanting, while it usually accompanies the nuclear. In many instances where toxins are concerned it seems as if they exhibited selective tendencies. One cause for the frequency of this selection, perhaps, can be ascribed to the fact that the ciliary muscle and its nerve supply have a highly differentiated organ. No doubt in the autotoxic type, though the lesion is frequently peripheral, yet the nucleus may be slightly involved. The same may be said of the purely toxic type.

By far the majority of the cases are peripheral paresis or paralysis, as only the act of accommodation is involved. If more than this is involved the chances are that we are dealing with some central or orbital lesion. Sufficient proof, however, has been given to show that the loss of accommodation can, at times, be a purely nuclear lesion without involving any of the other functions of the eye. Enough has been said to caution us not to neglect this apparent simple symptom in arriving at our diagnosis in such cases where little signs are of importance.

TREATMENT.

Little remains to be said about this. The indications are very obvious, namely, treat the underlying cause. For, a paresis or a paralysis of the ciliary muscle does not develop as such, but, whether peripheral or central, it has an underlying factor. The palliative treatment is the use of suitably adjusted lenses. The prescribing of eserine or strychnia may do some good, but the writer believes it is only apparent.

In general it may be said that the peripheral types offer better prognoses than the nuclear or central variety. The only caution to observe is not to mistake an asthenopia for a paresis or a paralysis.

The toxins of the various infectious diseases act on the peripheral mechanism of accommodation in much the same manner as the mydriatics or miotics. Finally, from what has been said, we notice that paresis or paralysis of the ciliary muscle as a peripheral symptom is quite common.

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DISCUSSION.

DR. D. S. REYNOLDS, Louisville, Ky.—I regard this as one of the most important papers I have ever heard read. It opens up for discussion a many-sided question that has at times great clinical significance. Without undertaking to look into the theories discussed by the essayist, I wish to say that I regret he had overlooked the fact that some people have the voluntary power of controlling the pupillary motion and the accommodative function. Roget, of London, whose *Treatise on Physiology* was published in 1838, was able to dilate his pupil at will and control the exercise of the accommodation. He could go on reading the finest type and dilate the pupil widely or contract it at will. Benj. Travers, a distinguished ophthalmic surgeon of London, published a letter from Dr. Roget, in a *Treatise on Ophthalmic Surgery*, published in 1820-4, before the time the *Physiology* of Roget was published.

I am obliged to take issue with Dr. Suker when he says that, in the paralysis of accommodation in diphtheritic cases, the prognosis is always favorable. I know of more than one case of paralysis of accommodation occurring in the course of diphtheritic disease that has remained persistently for years, and it has not disappeared. I know an instance more than 20 years old; I mean it is more than 20 years since the diphtheria occurred in a child of ten. Of course it may be argued that the diphtheritic poison was not the sole cause of the paralysis, and it would be difficult to refute the argument, that other causes may have existed, but the fact remains, as tending to show that, the prognosis is not always favorable.

I have seen two persons, with congenital absence of the iris; in one there is no accommodative power, and in the other normal range of accommodation exists. These are not to be forgotten nor overlooked. There are other cases in which absence of the function of accommodation exists, and which cannot be certainly accounted for.

I am very grateful to Dr. Suker for the time and care he has displayed in the preparation of this paper.

DR. ADOLF ALT, St. Louis.—While Dr. Reynolds mentions the fact that he has seen cases in which diphtheritic paralysis of the accommodation did not get better, I think the case he men-

tions as example was not an uncomplicated diphtheritic paralysis but probably some other disease was combined with the diphtheria. I have never seen such a paralysis that did not get well in the course of a number of weeks.

I beg to differ from Dr. Suker in his opinion that none but diphtheritic sore throats cause paralysis of accommodation. I have the records of a number of cases in which the bacteriological examination, the symptoms and the history of the case did not prove diphtheria, and in which a few weeks later paralysis of the accommodation occurred. I am satisfied that there are some infections of the tonsils and fauces that may cause paralysis of the accommodation which are not purely diphtheritic, or so little so that it is not possible to find the bacteriologic evidence and which, nevertheless, cause accommodative paralysis.

The Doctor recommends as treatment, palliative glasses. I do not see why we should give them. The accommodation assumes day by day a little more strength, and after a few days, you will have to get another glass, and at no time will it be of much value. I believe that the child who has diphtheritic paralysis should be carefully looked after, for fear of more important pareses and paralyzes supervening. I have the children abstain from eye work and do not allow them to go back to school as the family physician frequently does. I tell the parents to watch them very carefully, and especially not to let them run about. Sometimes to make them more comfortable I contract the muscle of accommodation by using eserine and usually give *nux vomica* or some such nerve stimulant.

DR. FRED. C. HOTZ, Chicago.—While listening to Dr. Suker's paper I was struck by the different tendencies that to-day two essayists have presented. Dr. Alt tried to separate two distinct pathological conditions which so frequently have traveled together. He showed that there is one group of episcleral inflammation in which the nodules appear upon the episclera, and another class of cases in which diffuse infiltration was present. If that distinction would be adopted, I think a good deal of discussion as to the merits and demerits of the treatment would be eliminated.

On the other hand, it seems to me, if I caught the drift of Dr. Suker's paper rightly, he applies the term of paresis and

paralysis to a number of cases or conditions in which it is not proper. He speaks of paresis of the accommodation following diseases that exhaust the system—typhoid fever and others. It is true that after an exhaustive disease most people cannot use their eyes for close work very long; if they are presbyopes, not at all; but the question is, is it proper to speak of such conditions as paralysis? Paresis means simply a low grade of paralysis. Those people are unable to keep their eyes converged very long. I suppose we should speak of insufficient converging power. Paralysis, if I understand the term right, refers to the nerve—to a reduced or limited supply of nerve force to the muscle. After such diseases the nerve is all right, it seems to me. It is the muscle which cannot act under the same nerve stimulus. The muscles cannot maintain the work required of them with the nerve force applied. These people cannot walk very long either, but certainly no one speaks of their legs being paralyzed or parietic; and that is the point I want to bring out, that this paper would lead you to speak of paralysis or paresis of accommodation under conditions where no such thing has existed.

DR. ADOLF ALT, St. Louis.—It has just occurred to me that I forgot that I wanted to refer to those cases in which there is a congenital insufficiency of accommodation. Here the muscle of accommodation is not sufficiently strong to do the full work required of it. Such cases surely would come under a separate class, and cannot be spoken of as parietic or paralytic cases; although their range of accommodation is smaller than it should be. These cases I think the author of the paper did not refer to.

DR. THOMAS FAITH, Chicago.—The doctor makes the statement that contraction of the pupil and accommodation are not associated actions, and while this may be true in a certain limited number of cases, it is not the case in the average normal eye, and our modern text books of physiology teach that they are associated actions.

Another point is the statement that the differential diagnosis between paresis and other affections of the accommodation can be made by taking the range of accommodation. This I do not believe, as the range of accommodation differs so much in the same individual at different times and in different individuals at the same time.

DR. WM. E. GAMBLE, Chicago.—The question of drugs producing paresis of accommodation has not been dwelt upon. I have lately had an experience which emphasizes the importance of the careful exclusion of drugs as an etiologic factor. A man came to me with paralysis of accommodation which had existed for several weeks. I went over the case carefully without finding any cause for the trouble. I, however, discovered he had been taking a drug for some weeks and asked him to return and bring to me his prescription. This showed that he had taken 1-96 of a grain of hydrobromate of hyocin every two hours. I advised him to discontinue it and in a few days the accommodation was better. I think we should inquire as to the drugs that have been taken in these cases.

DR. J. P. WORRELL, Terre Haute, Ind.—I have at the present time a lady patient who has a deficient range of accommodation on one side. She is unable to use her eyes for reading and sewing. I found the manifest hypermetropia was greater upon the side on which the near point was the more remote, but a careful study under mydriatics showed there was no difference in the refraction. When the total hypermetropia was corrected and the accommodation was restored the near point in the affected eye remained more remote than in the other eye. I have attempted to correct this disparity by giving a stronger glass for reading on that side. This plan has been successful in this case, but I would like to know what is the experience of the gentlemen in the use of reading glasses stronger on one side than the other in cases in which the refraction is the same. I feel like commending the views of Dr. Hotz as to the impropriety of calling this paralysis. The eye shows no evidence of paralytic changes. The condition may be one of diminished muscular power, or one of increased resistance to be overcome.

DR. H. H. BROWN, Chicago.—I have very much enjoyed the paper of Dr. Suker. I feel that Dr. Alt made a very important statement when he takes issue on the necessity of pharyngeal involvement. I have a case, a daughter of a physician, a characteristic case, which came to me with complete paresis of the accommodation. She had been in school constantly until the time of isolation made necessary by the out-break of diphtheria in the family, at which time an adult member of the family died.

The little girl was the picture of health and had no evidence of pharyngeal involvement. The paresis was as complete as any I have ever seen. The father being an intelligent physician, and the death of one member of the family giving every reason for care on his part, I am quite sure there was no pharyngeal involvement.

DR. EDWARD B. HECKEL, Pittsburg.—I wish to put myself on record as being of the same opinion as Dr. Alt, namely that a non-diphtheritic tonsilitis may be followed by a paralysis of accommodation. I have seen several cases where diphtheria could not be demonstrated. In seeking for a cause it is often difficult to arrive at a definite conclusion. In the use of drugs, as mentioned by Dr. Gamble, interesting problems arise. I once had a woman with a complete paralysis of accommodation without any apparent cause. She denied having used any drugs for a long time, but upon a closer inquiry I ascertained that she had been using a liniment on her husband. I obtained a copy of the prescription and found that it contained 2 dr. of fluid extract of Belladonna to a 3 oz. mixture. She had absorbed sufficient Belladonna through the skin of her hands to produce a paralysis of accommodation, which promptly disappeared after a discontinuance of the administration of the liniment. Paralysis not infrequently follows the use of a Belladonna plaster. I believe local paralyses are often due to a disturbance of the eliminative functions. In diabetics, I have seen a paralysis of the sixth nerve followed by a complete paralysis of all the third nerve muscles, and all of which made a complete recovery after the use of Carlsbad salt, which simply increased the eliminative functions and removed the toxic elements. The paralysis we see in hysteria, I believe are due to a faulty elimination, brought about by a physical disturbance of the normal functions and are toxic in character.

DR. ADOLF ALT.—Dr. Brown misunderstood me. I did not say I had ever seen paralysis of accommodation without pharyngeal involvement. I said that it is sometimes impossible to discover diphtheritic involvement in a sore throat. Yet a paralysis of the accommodation follows. Since the drug question has been brought up I wish to add an instance from my experience. A young gentleman rushed into my office with the exclamation:

"O, Doctor, help me, I am going blind suddenly. If you cannot, I shall kill myself." After I had quieted him, I found he had paralysis of the sphincter pupillæ and ciliary muscle in both eyes. Careful inquiry finally elicited the fact that he had used a patent suppository which contained Belladonna for the cure of hæmorrhoids. The recovery was, of course, a speedy one.

DR. REYNOLDS.—I would like Dr. Suker to give the distinction between *paresis*, which is central and *paralysis*, which is peripheral.

DR. SUKER (closing discussion).—I am not in the habit of taking issue with Dr. Brown, but I did say in my paper that it was not necessary to have diphtheria in the throat in order to have the paresis of accommodation, but that the diphtheria might be located anywhere. Although we might make 50 slides without demonstrating the bacillus, we still might have a mixed infection, or a diphtheritic involvement of very low grade. In such a case the bacillus might not assume its characteristic form. In reply to Dr. Hotz I will read a distinction I have made in a foot note, which will answer Dr. Reynolds at the same time. "Paralysis is the suspension or abolition of functional power, especially in the nervous system, in which case there is a temporary or permanent loss of the power of motion or sensation, or both, in the part supplied by the affected nerve. Paresis is an incomplete paralysis, especially when not associated with any demonstrable organic lesion—limited to motion and not to sensation." I said there were two varieties, myopathic and neuropathic. The myopathic can follow general muscular enfeeblement which usually happens in such diseases as typhoid, malaria, etc. In any muscle which is exhausted the effete materials accumulate and we have an exhaustion paresis. The nerve force may still be there, but the muscle incompetent to respond to the nerve impulse.

The reason our pupils are so near the nasal side is because we are creatures of evolution. If we live long enough we will find the pupil at the tip of the nose, as has once been said by my friend, Dr. Wood. The center or the muscle of accommodation is separate from the iris contraction center in the third nerve nucleus. Any one can by practice suspend the accommodation in one eye and maintain it in the other. My assistant has

noticed it in my own accommodation, while using the ophthalmoscope. The same may be said of the iris motility. The main fact is that we have a separate center for the muscle of accommodation and the muscle for iris contraction in the third nerve nucleus. It has been proven that they are separate.

As to palliative glasses, I merely wish to help out and comfort the patient for the time being, as it may be a business man whom I want to tide over his difficulty.

The symptoms of a peripheral or central paralysis or paresis are practically the same. To determine whether it is a central or a peripheral lesion depends mainly upon the fact whether the act of accommodation alone is involved or whether it is associated with mydriasis or the involvement of other muscles supplied by the third nerve. If more than the accommodation is involved, excluding mydriatics, the chances are that there is a central lesion, for the simple reason that all these centers are so intimately connected that in a nuclear lesion it would be nigh to impossible for one to be affected and not the other.

BLEPHARITIS MARGINALIS.

By DUDLEY S. REYNOLDS, A.M., M.D.,

LOUISVILLE, KY.

ONE of the commonest forms of blepharitis marginalis, seen in persons of every rank and station in life, and of all ages, is that in which there is slight thickening of the borders of the lids. The anterior margins are dry and red, and the lash, on close inspection, is observed to be thin, whilst the cuticle between the hairs is covered with minute scales of desquamating epidermis. This condition seems aggravated by constipated bowels, by the loss of sleep, and by fatigue of any kind. For convenience I will designate this class A. Cases of this kind are frequently observed to almost entirely disappear under correction of errors of refraction, of intestinal constipation, and of any form of mal-nutrition, or debility; whilst, on the recurrence of any of these abnormal conditions, or the use of the ametropic eyes without glasses, the local affection in the margin of the lids reappears. This is a condition that seems to be least susceptible to the action of any

of the mercurial ointments. In fact, the fungus present in the hair follicles seems incapable of producing much thickening and excoriation until some hyperæmia is set up, then the blepharitis takes on active symptoms. In cases seemingly cured by correcting errors of refraction, etc., close inspection will disclose a small, sheath-like elevation of epidermis around each hair in the lash. I believe the fungus in the follicles in such cases may be entirely eradicated, and permanent recovery secured for many of them, by the periodical application of pure carbolic acid. I apply it with a needle, first preparing the acid by adding about ten minims of alcohol to the drachm of crystalized acid, and agitating sufficiently to dissolve all crystals. This will usually remain in liquid form as long as it is kept in a well-stopped vessel. Into this fluid I dip the point of the needle, and, holding the lid firmly with the fingers of the other hand, I scrape off all the detachable scales between the lash, making the application as nearly direct and complete to all the hair follicles of the margin of the lid as possible. By repeatedly dipping the needle into the acid, and scraping the skin, taking care that none shall be allowed to run over the free border, the application can be made so thorough and complete as to whiten all that portion of the skin containing the lash. This creates a little temporary smarting, which is not severe, and which lasts not more than two or three minutes. About the fifth day after each of these treatments a crust of epidermis exfoliates, and should be removed, and a little yellow oxide of mercury ointment applied. The application of the carbolic acid should be repeated about once in ten days, in ordinary cases. In the course of about three months, it will be observed in most cases, especially in young persons, that a heavy and luxurious growth of lash has come on, and with it entire disappearance of all the manifestations of blepharitis. I have observed that most of these cases seem permanently cured, after a lapse of five or ten years.

As an illustration of the intractable nature of this disease, I invite attention to the case of Miss D., aged 13, who came to me August 22, 1899. The margins of the lids of both eyes were slightly thicker than normal. On close inspection it was observed that the anterior margins were covered with fine scales of desquamating epidermis. There were no signs of hair where the lash should be in either of the upper lids,

and but a few fine, short hairs along the margins of the lower lids, and these were scattered at long intervals. She had for two years been under the constant treatment of eminent specialists, and had been told by one of my ablest confreres that she would never again have any lash. The family were anxious, and I felt the necessity of being cautious about making a prognosis. I asked for two weeks time in which to form an opinion as to the probability of a re-growth of lash. I applied the phenol in the manner described, to the right upper and lower lids. She was unwilling to have the application made to the other eye at this time, so we had to wait; meantime using the yellow oxide of mercury ointment to the left eye, with friction, once every day. August 25th the crust exfoliated from the lids of the right eye, and the yellow oxide or mercury was applied, with friction, with the edge of a Daviel spoon. At the end of a week from the date of the first treatment a magnifying glass disclosed evidences of the return of a few lashes, and I made an encouraging prognosis as to the right eye. The left, which showed no signs of improvement, was then subjected to similar treatment.

During the fall I was permitted to apply one more treatment of this kind to each eye, and before Christmas she had a good growth of rather heavy lashes, with almost total disappearance of the itching of the lids and roughness of cuticle, which had greatly annoyed her for months before she came to me.

In the spring of 1890 I did not see her. About the first of June the lash suddenly fell out. She returned, and at intervals during the summer of 1900 she received treatment with the phenol, and soon enjoyed a luxurious growth of lash, which was the envy of all the girls in her school.

In September, 1901, it was thought the upper lash looked thin, so I applied the phenol to both eyes. The lash became more luxuriant, and no further trouble came until September, 1902, when, the patient having gone to Cincinnati to live, wrote me her lashes had again fallen out. I referred her to Dr. Derrick T. Vail, and do not know how she is since.

(B.) The cases which present an excoriated, glazed, red, rounded appearance of the tarsal margin, with no sign of lash, are seldom seen with both eyes affected to the same degree. It often happens that one eye may be placed in class A, whilst

the other presents an aggravated condition, with a tendency to fissure at the external canthus. Constitutional disturbances and errors of refraction are alike provocative of aggravated symptoms in these cases. I have never observed much relief from local treatment with ointments; and, for that matter, nothing approximating curative results from any sort of treatment, excepting the phenol, applied as described in class A. I have frequently observed, where the lid is very greatly thickened, the margin rounded off, and of a bright red, glazed appearance, absolutely free from any sign of lash, after two or three applications of the phenol, at intervals of ten days, great reduction in the thickening of the lid is secured, and a fair growth of lash begins to appear; and, by the continued prosecution of this plan of treatment, with constitutional correctives, absolutely complete recovery occurs in many persons under forty years of age. It is especially efficacious in children, who are always averse to systematic rules for applying ointments.

(C.) Another class of cases is that in which an abundant accumulation of inspissated sebum mats the lash together in groups. When the crust is removed it is usual for most of the hairs of the lash to come away with it, disclosing an ulcerated condition of the lid, which destroys the hair follicles. This condition is sometimes associated with great thickening of the margin of the lid, and dilated veins are often seen coursing over the surface of the lid, just under the skin. On everting the lid, a relaxed and flabby condition of the retro-tarsal conjunctiva appears. There is profuse lachrymation, and sometimes photophobia. The crust is best removed, after an application of simple petrolatum, with a cotton mop, made by rolling a bit of cotton on the end of a probe, and dipping it into the ointment, and smearing the lids in contact with the upper boundary of the lash. After a little rubbing with this mop, the crust becomes loosened at the upper edge and may be lifted off with the dressing forceps, or turned out with the end of a small probe. The ulcer, which is sometimes deep and of a conical form, should now be cleansed by pressing into it a clean cotton mop; and, after removing all the moisture, a small portion of phenol should be applied to the bottom of the ulcer. Keeping the lid separated to prevent any flow of tears carrying the phenol between the lids, it will

soon be observed that the whitening produced by the phenol has disappeared by becoming incorporated with the tissues. The patient may now have a piece of gauze, smeared with petrolatum, laid over the closed lids, and a bit of cotton wool on the outside of the gauze, secured with a few strips of plaster, laid from the cheek to the forehead. This dressing should not be disturbed for twenty-four hours, when the treatment may be repeated, if necessary, as at first, excepting that it will be observed no crust has reappeared in the lash, and therefore no preliminary treatment is required. The ulcer, however, should be carefully dried and again treated to an application of the phenol. In some cases a single treatment will be found sufficient to cure the ulceration. By this plan of treatment some of the worst cases may be entirely cured within ten days. Always bear in mind, however, the necessity for close attention to constitutional correctives.

There are many cases of ulcerative blepharitis marginalis that are undoubtedly due to inherited syphilis, and no plan of local treatment, unassociated with constitutional measures, will be found availing. The syrup of hydriodic acid is a priceless remedy in such cases, and may be given with the food to children of all ages, without risk of disturbing the gastro-intestinal system.

It should not be forgotten that most of the ulcers in this class of cases are broken-down gummatous formations. It is sometimes seen in the ciliary margin of the lids, and sometimes in the meibomian margin. In this class of cases the ulcer should be thoroughly dried, and the ulcer filled with a portion of yellow oxide of mercury powder, or a crystal of the red oxide.

There are cases, however, in which a minute pustule occupies a single hair follicle, or several contiguous follicles, but these cases are so distinct in appearance and so easily recognized by the unaided eye, as to make the diagnosis certain. It is often seen that a single hair is surrounded by a yellowish white opaque substance, which, when the hair is pulled out, is found adhering to it. Fortunately, this infectious folliculitis of the ciliary margin of the eyelid is not very common, as it invariably destroys the hair bulb in each infected follicle. Bathing the eye in a solution of chloride of sodium, fifteen grains to the ounce of water, quickly terminates this infection.

There are many complicated forms of ulcerated blepharitis marginalis, each of which require distinct modifications of local treatment, and so I do not wish to be understood as claiming that the local application of phenol is to be indiscriminatingly made. It is certain it could not suffice in cases of tarso-adenitis; nor in those cases where the cicatricial contractions from previous ulceration may have obliterated the hair follicles. It would be useless in mal-position of the puncta lacrimalia, nor would it be found sufficient in cases complicated with phlyctenular disease.

The condition in which alopecia is present, without ulceration or apparent desquamating conditions, or which may be due to parasitic or microphitic causes, in persons who have neither syphilis nor leprosy, the application of phenol often produces brilliant results. I have never seen hair restored in a case of palpebral alopecia, the subject of general alopecia. In syphilitic subjects, where the hair has fallen from the brow, eyelids and head, all efforts at restoring it to the lids by local applications have failed in my hands, nor have I seen the lash reappear in syphilitic cases under any form of constitutional treatment.

Not attempting to exhaust the subject, I have endeavored to portray a few well-known conditions wherein the modifications of local treatment I have suggested seem more advantageous than the methods hitherto employed or commended by our standard authors.

DISCUSSION.

DR. VAIL.—Since my name has been mentioned in Dr. Reynolds' interesting paper, I shall report concerning the case referred to and which Dr. Reynolds kindly sent to me. The girl came in, presenting a letter of introduction. I saw the lashes had fallen from the left eye. At a glance there seemed to be no inflammation at all, but on close inspection I recognized there was some exfoliation of the epithelium, and on drawing out one or two of the few remaining hairs, I found the characteristic black-rooted and club-shaped appearance we have all seen in diseased lashes. I sterilized the margin of the lid with H_2O_2 and applied 20 per cent. silver nitrate by means of a cotton tipped probe. The lashes promptly returned. At this time the young lady has the

beautiful drooping lashes described by the writers of novels. The healthy appearance remains to the present day.

I have been able to recognize clinically several different types of blepharitis, all of which are manifested in both the acute and chronic forms: (1) simple, (2) pustular, (3) eczematous, (4) membranous (which is exceedingly violent; I have seen a membrane like diphtheria in the lashes, in most cases failed to find the germ, although in one case I did find the Klebs-Loeffler bacillus), (5) deciduous (to which the case reported by Dr. Reynolds belongs), (6) ulcerated, (7) tineal (Lousey). The same remedies cannot be used successfully in all cases.

DR. SUKER.—I wish to find fault with the term “marginal blepharitis.” The name “blepharitis eczematosa” would cover the ground more fully. The loss of the lashes and thickening of the tarsus are secondary considerations. I am glad the author said not every case is due to refractive error, as we almost had to believe according to Roosa (1876), although I think many cases are hypermetropic. I am in the habit of using silver nitrate, 20 to 30 per cent., the same as the author uses the phenol, and with good results. The removal of the little coverings along the lashes, upon which the doctor laid stress, is a good point, though often neglected. In some cases ichthyol ointment acts admirably.

DR. HECKEL.—It seems to me we might eliminate the word “marginalis” altogether. The word “blepharitis” means an inflammation of the *lid*, but by common consent and usage it is limited to an inflammation at the margin of the lid, hence the modifying adjective “marginalis” is entirely superfluous. I think Dr. Suker misquoted himself when he said that he uses silver nitrate in a solution from 20 to 30 per cent. Did he not mean 20 to 30 grains to the ounce?

DR. ALT.—I am in the habit of using corrosive sublimate in the same way as Dr. Reynolds uses the phenol, and am very much pleased with the results. Having cleansed and treated the lid margins with the bichloride of mercury, I apply an ointment for protection.

DR. HOTZ.—I fully agree with Dr. Reynolds that the use of ointments in the ulcerative forms is useless. In that form the application of nitrate of silver 10 to 20 per cent. or more, or carbolic acid as described, gives the best results.

I was surprised to hear such a poor opinion about the efficacy of ointment in that mild form, so-called squamous blepharitis. I found the careful use of ointment of yellow oxide of mercury or ointment of ichthyol and vaseline bring good results, if properly applied. The patient cannot do it himself. The ointment in the hands of the patient will never relieve his trouble, because it is impossible for him to make the application properly along the upper lid margin. It would be very difficult for me to do it myself to my own eye. You must instruct some one in the proper application of the ointment to the patient's eye. In the case of children, show the mother; if adults, show some other person, or have them come to you and do it yourself. I have made the experiment so frequently that I am satisfied it is the way the ointments are applied that makes the difference in the results. I have had them come back in a week or two without change in the condition, and then I applied it myself twice, perhaps, and there was such a manifest change in the condition that the patient himself expressed relief.

DR. J. W. MURPHY, Cincinnati.—I think Dr. Heckel is wrong when he says it should be 20 to 30 grains of nitrate of silver to the ounce, in these cases. I use 20 to 30 per cent. silver. It is necessary to use this strong solution; after carefully cleansing the lid margin, with its use you thoroughly destroy the ulcer, and a healthy result goes forward. With the weaker solutions, 4 or 5 per cent., you will not have success.

DR. DAYTON.—I have a case in mind that might possibly account for the loss of those beautiful lashes. A girl of about the same age as Dr. Reynolds' case presented herself for treatment. I made a snap-shot diagnosis of blepharitis marginalis, but inasmuch as there was no perceptible inflammation present along the margin and no ulcers, I made an examination and found no disease of any of the follicles so far as I could see. I gave the yellow ointment with a very small amount of citrine ointment with it. She reported in a few days with no different appearance. I found on close questioning that she was apt to pull out hairs occasionally. I had her watched carefully for some time and not allowed to sleep by herself or be alone at all, and soon she had a very fine growth of lashes. She went on a visit away from home,

but this visit was brought to an abrupt termination because of the renewed loss of the lashes. She was again put under surveillance, without treatment, and the result was a return of the lashes. One day I took her privately and gave her a very good talking to, and she confessed to me that she had pulled out her lashes. It was a hysterical manifestation at the age of puberty and she had deliberately plucked every lash as it appeared in the upper lid. Perhaps the author's case is of the same character.

DR. VAIL.—This matter of an hysterical element was thoroughly gone over in the case reported. I sent for the mother, as I suspected hysteria, and explained to her in a kind way that it was possible that this young girl—who did not seem to be hysterical but unusually sensible about everything—might possibly be pulling out her lashes, and requested that she watch her closely. She never lost sight of her for a week and came back with the patient, declaring that there had been absolutely no interference. The lashes simply came away without any resistance and with none of the evidence of pain a patient gives when you draw a healthy lash. I am certain that this is not an hysterical case. I have met such cases, but do not believe this is one.

DR. M. D. STEVENSON, Akron, O.—When the borders of the lids are diseased it is necessary first to cleanse them thoroughly, removing all crusts and scales when present in order to learn their exact condition. Frequently the cleansed lids are found to be merely reddened (a vascular condition usually due to asthenopia) and not truly inflamed, the crusts and scales being the result of hypersecretion of the local glands and desquamation of epithelial cells. Many lids are found to be truly inflamed and some to have little pustules at the roots of the lashes involving the sebaceous glands. These particular lashes should be epilated and the ulcers touched with a 20 to 30 per cent. silver nitrate solution. A mild, clean ointment kept in collapsible tubes, applied by a clean finger to the cleansed lids, is of great service, softening and at the same time facilitating the removal of the scales and crusts, keeping the orifices of the local glands patent and protecting the skin from the tears. Too often ointments are prescribed in common jars, allowing them to become dirty and rancid,

and the patient is not sufficiently impressed with the need of cleanliness in their application. A dirty, rancid ointment applied by foul fingers to unclean lids, is more productive of harm than good.

DR. REYNOLDS (closing discussion).—I am thankful for the discussion the gentlemen have bestowed upon my feeble effort. As to the classification, I confess it was not meant to be scientific, but rather commonplace and clinical.

As to the possibilities of hysteria in the case I referred to Dr. Vail, I used the magnifying glass in the inspection of the ciliary margin of the lid, and finding no young lashes present, as would be the case had they been pulled out by force, I eliminated in that way the possibility of hysteria as an ætiological element. If the lash is plucked out with the finger nails, it is impossible to get all the young hairs. It is not possible for the patient to grasp them with the nails. The lid was absolutely bare, in this case, a complete madarosis.

As to the nitrate of silver, bichloride of mercury or any other form of caustic, I can conceive of no material difference in the result. The difference is simply as to preference on account of pain in the one case and its absence in the other. While the chemist may tell you that a little alcohol destroys carbolic acid, mixed as I mix it, it answers every purpose. I should hate very much to swallow alcohol containing carbolic acid in solution under the impression it had been destroyed. It is like a good many other things that chemistry teaches us—it may be hypothetically true, but in fact it is not true.

As for the treatment of any form of blepharitis with ointments, I fully concur with the gentleman who says it is necessary for the surgeon to apply them. The mother of this young lady was trained how to apply the ointment by means of a bit of cotton rolled on a stiff wire. As to the use of the solutions of nitrate of silver, I see difficulty in preventing this running over the border of the lid. The carbolic acid can be applied to a restricted area without risk of spreading. The pain it produces is very light and transitory, whilst silver and mercury produce burning pain which persists for an hour or more.

A SERIES OF GLAUCOMA CASES.

By DR. GEORGE F. FISKE.

CHICAGO, ILL.

No.	Name	Sex	Age	Eye	Refraction	Diagnosis and Operation.	Result.
1	A. G.....	M	49	O. U.	+2 D.sph	Iridectomy O. U.	V= $\frac{20}{30}$, no recurrence.
2	Mrs. L. C.....	F	55	O. S.		Traumatic glaucoma; Evisceratio bulbi.....	Right eye remains normal.
3	Mr. W.....	M	48	O. U.	+3 D.	Irid. O. U.....	No recurrence.
4	H. B.....	M	41	O. D.	Em.	O. D. acute glaucoma, iridectomy.....	V= $\frac{20}{30}$, no recurrence.
5	Mrs. M. A. Mc	F	76	O. U.	Hyp. ast.	O. U. glaucoma simplex, O. U. Irid.....	No recurrence.
6	Mrs. M. R....	F	70	O. D.	?	O. D. hæm. glau. irid. and evis. bulbi.....	Left eye remains normal.
7	Mrs. E. F.....	F		O. D.	+4 D.	Glau. acute irid.	No recurrence.
8	A. J. B.....	M	43	O. D.		Glau. chron. irid. 3 times and evis.	O. S. remains normal.
9	A. S.....	M	60	O. D.		Glau. simplex chron. irid., later evis.....	O. S. remains normal.
10	Mrs. M. S.....	F		O. U.	+150	Glau. chron. irid. O. U.....	O. D. $\frac{20}{60}$, no recurrence.
11	Mrs. M. W....	F		O. U.		Glau. chron. O. U. irid.....	Fingers at 2-4 feet.
12	Mrs. G. W....	F	47	O. S.	+250 D.	Glau. chron. irid. O. S.....	O. D. remains normal.
13	Mrs. W. G....	F	60	O. U.	+150 D.	Glau. chron. O. U. irid.....	O. S. V. = $\frac{4}{30}$.
14	Mrs. B.....	M		O. D.		Glau. chron. irid. O. D.....	O. S. remains normal.
15	Mrs. W. J. W.	F	34	O. U.		Glau. chron. O. D. evis. O. S. irid.....	O. S. fingers at 1-2 feet.
16	A. D.....	M		O. S.	+150 D.	Glau. acute O. S. irid.....	Right eye normal, O. S. $\frac{5}{6}$, no recurrence.
17	W. C. P.....	M	58	O. D.	Hyp.	Glau. chron. O. D. irid.....	O. S. normal.
18	Mrs. D.....	F	60	O. U.		Glau. acute O. U. irid.....	No recurrence, V. = $\frac{20}{40}$.
19	Mrs. E. P. B.	F	63	O. D.	+450	Glau. chron. O. D. irid.....	O. S. remains normal.
20	J. F. W.....	M		O. D.	Em.	Glau. acute O. D. irid.....	V. = $\frac{20}{30}$, No recurrence.
21	Mrs. L. Mc.	F	73	O. D.		Glau. chron. irid. by Dr. Knapp.....	Eye blind, no recurrence.
22	B. C. D.....	M	39	O. U.	+450 D.	Glau. sub acute O. U. irid.....	V. = $\frac{5}{6}$, no recurrence.
23	Miss M. K....	F	30	O. D.	+1 D.	Glau. chron. O. D. irid.....	No vision, O. S. remains normal.
24	Mrs. J. T.....	F	42	O. D.	+1 D.	Glau. chron. O. D. irid. twice.....	O. D. no vision. O. S. Irid. advised.

No.	Name	Sex	Age	Eye	Refraction	Diagnosis and Operation	Result
25	W. M. E.....	M	60	O. S.	+1 D. cyl	O. S. Glau. acute irid. O. D. Glau. acute O. D. irid. 4 yrs later	O. S. V.= $\frac{5}{6}$. O. D. V.= $\frac{20}{40}$.
26	D. J. H.....	M	60	O. S.		Glau. acute O. S. irid.	No recurrence O. S., O. D. blind years ago following Gl.
27	H. A. O.....	M	53	O. S.	+1 D.	Glau. subacute O. S. irid.	V.= $\frac{5}{10}$, operation advised, O. D. later.
28	Mrs. H. B. A.	F	66	O. S.	+2 D.	Glau. simplex irid.	No recurrence V.= $\frac{20}{120}$.
29	J. H. W.....	M	58	O. S.	+150 D.	Glau. hæm. irid. and evis.	O. D. remains normal.
30	Mrs. J. H. E.	F	30	O. S.		Glau. acute slight attack, no operation	Eye normal.
31	W. H. J.....	M	39	O. U.	+1 D.	O. D. glau. acute O. S. glau. chron. O. U. irid.	O. D. V.= $\frac{5}{5}$. O. S. V.= $\frac{20}{80}$.
32	Mrs. L. B....	F	66	O. S.		Glau. chron. O. S. irid.	No central vision O. D. remains normal.
33	R. P. F.....	M	58	O. U.	Hyp.	Glau. acute O. U. irid.	V.= $\frac{20}{80}$, no recurrence.
34	J. B.....	M	62	O. D.		Glau. acute irid.	V.= $\frac{20}{40}$.
35	N. S. H.....	M				Glau. chron. irid.	O. U. blind, evis. may be necessary.

THESE thirty-six cases have been collated for the sake of making a few deductions, more or less authorized, and bringing up for discussion some practical questions connected with this disease, which is certainly to be dreaded by all of us.

As I understand it, the ætiology of glaucoma is still, after a great amount of research and discussion, by no means satisfactorily settled. It is perhaps fair to say that the symptoms can be best explained as due to an increase of tension, in its turn due to difficulty of excretion as compared with secretion. This being granted, the point to determine is the cause of this difficulty of excretion. In these cases of mine, there is not a single one of glaucoma in a myopic eye, and almost all the eyes are hyperopic. This corresponds with observations of other ophthalmologists, and careful measurements made by other observers all point to the small eye as being the one peculiarly liable to the disease. It seems to me, then, that the best explanation is that of Weber and Priestly Smith, which attributes the difficulty of excretion to the small eye and lens of normal size, that is, large in proportion.

In these eyes the ciliary body is enlarged and the flow of the lymph from the vitreous into the posterior chamber is hindered. When the veins of the ciliary body become too full, the swelling cuts off communication between the vitreous and the posterior chamber and the ciliary body is pushed forward and presses the root of the iris against the sclera at the point of union with the cornea.

This question of the ætiology I have gone into at some length because it brings us logically to the question of what to do in glaucoma, and I believe in and wish to advocate the early iridectomy in all cases of glaucoma; this iridectomy to be followed by a second and third, if necessary, immediately the symptoms return.

It may seem an apology is due you for bringing up an old matter, and you may say, "We all of us believe in and practice iridectomy for our glaucoma cases." My experience has been, however, that many of our best ophthalmologists resort to massage, and eserine, and heat, and procrastination with their patients in the early stages, at the time of the first attack; and it is right here that the operation is most useful and most likely to conserve the sight.

Then comes the question as to the operation upon the second eye. As we all know, in the majority of cases the second eye is sooner or later affected by the disease. I remember hearing a brilliant paper on this subject where it was advised that an iridectomy should be performed upon the second eye, even when sound and when no symptoms of the disease had presented themselves. Personally I believe that it is quite demonstrable that such a course would save many from blindness, though I doubt if any of you will here advocate such a procedure, and this is one of the questions I wish to bring up for discussion to-day—and for earnest discussion, recognizing the fact of the danger of an iridectomy injuring a sound eye, and on the other hand not forgetting the cases where an iridectomy should have been performed at the time of the first, or one of the earlier attacks, and where it has been put off by patient, or surgeon, or both, and the sight has been lost.

In looking over the cases in this paper there is one where there was an acute attack in one eye, which was slight, and

where no operation was performed, and where up to this winter there has been no recurrence.

In this list there are sixteen cases where up to the present time the second eye has not been affected. There have been five eyes where evisceration has been necessary, and there are two more eyes where it may be necessary later. All these seven eyes were affected with the disease for some time before iridectomy was performed. The experience in these cases was that sight was preserved and the disease checked just in proportion as the iridectomy was early.

In operating I used a cataract knife and make a large incision just beyond the edge of the cornea, not trying to go far into the sclera. Then I remove a large section of the iris and reach its root so far as possible. I use a two per cent. solution of eserine after the operation and prepare for the operation by the use of eserine and hot compresses if the tension is great, and use bichloride 1 to 5,000 as antiseptic.

CASE 4. H. B., age 41, seen Sept. 12, 1887, with typical case of acute glaucoma, marked by vomiting, very high tension, great pain. Entire absence of vision in the right eye and grey cornea. Iridectomy September 14th without hæmorrhage under ether, and eye treated with eserine and hot fomentations. The pain persisted and iris seemed sluggish, the conjunctiva remaining bloodshot, with no tension. A two per cent. solution of atropine was used twice a day, under which treatment the eye improved with great rapidity.

Sept. 24. V. = $\frac{20}{20}$.

Jan., 1889. V. = $\frac{20}{20}$.

No recurrence in either eye.

5. Mrs. M. A. Mc., age 76. Seen first March 5, 1889. Diagnosis: glaucoma simplex of a year's standing in right eye. Uncertain in left, but failure in vision marked within last few weeks. Diagnosis corroborated by three other oculists, two of whom advised against operation, while one agreed with me that the operation afforded the only chance.

V. O. D. +1 D. sph. \bigcirc +1.50 D. cyl. $180^{\circ} \frac{10}{200}$.

O. S. +1.50 D. cyl. $180^{\circ} \frac{20}{80}$.

O. U. Incipient cataract. Excavation marked. Field characteristic, no pain or tension. O. U. iridectomy downward March 9, 1889.

May 20. $V \odot + 2$ D. cyl. $180^\circ \frac{5}{6}$. O. D. fingers at 5 ft.

Dec. 2. O. S.—0. 75 D. sph. $\odot + 1.50$ D. cyl. $180^\circ \frac{5}{6}$.

Later the vision failed slowly in the right eye and remained in statu quo in the left eye. June 6, 1893, O. S.—2 D. cyl. $75^\circ \frac{5}{6}$. The patient died soon afterward, able to read up to the time of death. This case is dwelt upon at length, because it was very unfavorable with regard to prognosis, in the absence of acute symptoms.

6. Mrs. M. R. B., age 70. Seen July 6, 1889. O. D. acute attack of glaucoma supervening upon an old case dating back years. Great pain. $T+2$. Anterior chamber shallow. Pupil dilated, immovable. Fingers not counted. July 8 iridectomy, followed during the night by hæmorrhage and pain. July 17, evisceratio bulbi. The refraction of the left eye was -0.50 D. sph. $\odot - 1$ D. cyl. $90^\circ \frac{5}{6}$, and the sight has remained good up to the present date. The refraction of the right eye could not be determined.

No. 8. Mr. A. J. B., age 43. Seen May 1, 1890. O. D. vision = fingers at 2 ft. Shows typical glaucoma excavation. Was operated upon twice several years before, iridectomies, though vision had disappeared previous to these operations, as he had been treated for a year with pills. No. 1 and 3, taken twice a day upon advice of a homoeopathic oculist. O. S. $V \odot - 1$ D. cyl. $88^\circ \frac{5}{6}$. In 1893 I eviscerated the right eye because of the unbearable pain. Left eye is in good condition today.

No. 36. Mr. K., age about 32. Suffered from acute glaucoma in both eyes. Iridectomy performed in Halle several times on each eye, so that almost the whole iris was removed. Sclerotomy was also performed twice. This was one of the most remarkable cases I have ever seen, because of the persistent return of the glaucoma and because of its eventual yielding to repeated operations. I saw the patient first in 1883 in Prof. Alfred Graefe's private clinic in Halle, and last in my office in Chicago in 1894 or 5, at which time the sight was very good in both eyes and there had been no symptoms of glaucoma for years.

No. 35. Mr. N. S. H., age 40. O. N. Blind for past three weeks, due to glaucoma of two to three years' standing. This man had never consulted an oculist, believing that the

failing sight was due to his chronic kidney disease. O. U. T+2. Iridectomy to relieve the intense pain. No vision of course resulted, and while the pain is now absent, after a lapse of three months, it is very possible that the eyes may have to be enucleated later.

DISCUSSION.

DR. W. K. ROGERS, Columbus, O.—This subject can scarcely fail to be of the most intense interest to all of us, and I am sure no apology is called for in behalf of a paper on this subject, in connection with which so much has been written and done, and concerning which the views of many observers have undergone considerable change in the past few years. The point of principal interest to me in the paper is the allusion to operative interference in cases of a chronic character, and I presume the doctor refers to the simple, non-irritative variety of glaucoma under this head. I believe there is a growing tendency to resort to iridectomy in these cases, as compared with the past, and I would like to add my small modicum of experience. I have operated on seven of these cases, covering a period of ten years, and I have not had cause to regret this procedure in more than one instance, and even in that it is fair to assume the difficulties experienced might have been encountered even without the operation. In this case the patient was a woman upwards of 65 years of age, with marked atheromatous condition of the vessels. It was necessary to use a general anæsthetic, and the patient developed an attack of emphysema with paroxysms of coughing, which resulted in intra-ocular hæmorrhages. She eventually made a fair recovery. One eye retains vision practically equal to that before the operation, and the other eye suffered a diminution of about two-thirds. This was some seven years ago, and up to the present time there has been no increase in the impairment of vision and no exacerbation of the glaucomatous symptoms, whereas before operation vision was progressively deteriorating.

DR. W. H. WILDER, Chicago.—I want to compliment Dr. Fiske on his excellent report of cases. It is certainly a most important subject and one, it seems to me, which can not be covered as thoroughly as it should be in the time at our disposal. I think very few of us doubt the efficacy of

iridectomy in the acute or even the chronic forms of glaucoma where there are acute attacks. As to the pathology, I do not see how the theory of Weber and Knies holds good when the anterior chamber is deeper than normal. In such cases I prefer the anterior sclerotomy or the posterior sclerotomy. I had a case with brilliant results where the posterior sclerotomy changed the vision from almost zero to $20/20$ in two weeks, and this good condition remained for two or three years, before the case passed out of my observation. It seems to me the most important thing in the study of glaucoma and the study of the effect of operations, is a careful record of the field of vision. Every one has had cases of chronic glaucoma where central vision was normal, as in some of Dr. Fiske's cases, and where the peripheral vision is much contracted. I have a most unfortunate case, in which the man's central vision was $20/40$ up to the day I did an iridectomy. He had absolute glaucoma in the left eye. In the right eye slight increase of tension, excavation of the optic disc and $20/30$ of vision with a marked contraction of the field. The field contracted more and more until it seemed to him as if he were looking through a gun barrel. Still he had $20/40$ and better of central vision. An iridectomy was done, and immediately after the excision of the iris the vitreous was forced into the wound and this was quickly followed by a profuse intra-ocular hæmorrhage.

DR. C. BARCK, St. Louis, Mo.—This subject is always an interesting one, especially as the opinions are still divergent as to the best method of treatment. Until lately many, especially American ophthalmologists, were opposed to operative interference in chronic glaucoma; but late years have furnished us statistics which enable us to judge better on the final results of operations. One of them is from Hirschberg's clinic in Berlin (*Centrbl. f. Augenhk.*), the other is by Haab, in Zürich (*Glaucoma and its Treatment*, 1902). Both authors took especial care to keep the cases under observation—which is easy in countries where the population does not often move from place to place—and only such cases were included in the statistics in which two or more years had elapsed since the operation. The statistics agree with each other to a remarkable extent. Haab's results were as follows:

Glaucoma simplex, 76 cases—resulted in blindness, 29 per cent.; fair result, 29 per cent., good result, 42 per cent. =71 per cent.

Glaucoma, inflammatory, chronic, 37 cases—resulted in blindness, 43 per cent.; fair result, 27 per cent.; good result, 30 per cent.=57 per cent.

On the other hand, out of the 15 eyes treated with drugs alone, the results were, blindness, 60 per cent.; fair result, 40 per cent.

Hirschberg's results were very similar.

In the light of these statistics, it seems to me that we cannot reject operative interference any longer in a disease with such a poor prognosis otherwise.

It seems to me that the different substitutes for the old-fashioned iridectomy, viz., anterior sclerotomy, incision of the iris angle, etc., have been gradually abandoned. My custom is, and I would advise, an iridectomy not only in acute, but also in simple and chronic glaucoma, as early as possible. This I follow up by the use of eserine or pilocarpine. Should the diminution of sight continue in spite of the first operation, I add a posterior sclerotomy.

DR. REYNOLDS.—I do not wish to discuss the theories and etiology of glaucoma. I concur with Dr. Wilder in the great importance of making frequent record of the field of vision. It is in the peripheral contraction of the field of vision that we find the first manifestation of increasing danger. I believe it is safer to do an iridectomy in all cases, because of the risk of an advancing peripheral contraction of the field and increasing amblyopia, which may be so nearly imperceptible as to deceive us.

I think it might be stated that one-half of the fatal results of iridectomies done in cases of high tension might be avoided by giving a dose of Rochelle salts, and for a few hours, after the first action of the salts, the salicylate of sodium in the definite dose of ten grains every half hour in a half pint of water, until pain disappears and tension is reduced.

At the time of operation a few drops of 1—1,000 of the chloride of adrenaline solution is important. This should be repeated at short intervals for the next ensuing twenty-four hours after operation, to restrain hæmorrhage.

DR. J. A. L. BRADFIELD, La Crosse, Wis.—I wish to report a case which illustrates the possibilities in what seems an almost hopeless case. In 1896 I was consulted by a stationary engineer, 35 years of age, for failing vision of right eye, which equalled $20/60$. Diagnosis: chronic glaucoma; iridectomy advised and rejected. Eserine was prescribed, but as I did not see him but once after, I do not know how long it was used.

July, 1901, he again consulted me. The right eye had been blind several months. Two weeks previous left eye became very red and painful. Patient vomited and had to be kept under the influence of morphine. As the vision became so poor he was unable to see even enough to recognize his own family, he again called on me, when I found the following condition: Vision of right eye—perception of light, tension plus 3; typical atrophic cupping disc. Left eye—severe ciliary injection; widely dilated pupil; cornea very cloudy; anterior chamber almost obliterated, and tension very high; pain, intense; vision, perception of moving objects.

Under holocaine anæsthesia made small incision with a very fine Graefe knife. The iris prolapsed with the completion of the incision and the wound gapped so as to greatly endanger the position of the lens.

Recovery uneventful. Four weeks after operation patient resumed his former work which he has followed ever since with correction of error of refraction. Vision equals $20/20$. Tension is perfectly normal and eyes comfortable.

DR. J. O. STILLSON, Indianapolis, Ind.—I rise to subscribe to what Dr. Barck has said as to the advisability of an early iridectomy. I do not feel that we understand the etiology of glaucoma. I usually find that in this early stage of glaucoma simplex there is a narrow anterior chamber. Whether it be the pressure of the proportionally large lens and small eyeball on the ciliary body, which causes the stoppage of circulation of the fluids from the posterior to the anterior portion of the eye, or not, we do have this condition here which evidently necessitates surgical interference. Even if the sight is good, I think we should urge an early iridectomy. I think that eyes are frequently lost in an effort to make an iridectomy in a narrow anterior chamber with a lance-shaped knife.

DR. HORTZ.—I think we will not find a satisfactory theory of glaucoma as long as we try to apply the same explanation to different conditions. Clinically, we have two different conditions—the typical glaucoma and the so-called glaucoma simplex. In the one we have periodical attacks or exacerbations and remissions. In the so-called simple glaucoma we have nothing of that sort. In the first class we have a shallow anterior chamber; in the second class we have a normal and sometimes even deeper than normal anterior chamber. In the first class we have tension increased; in the second class we often cannot find increased tension at all if we examine the eye every day. But we sometimes find a deep excavation of the optic nerve. This shows there must be some different pathological process underlying these cases. As long as we try to bring the two under one head we will not come to a satisfactory conclusion. The pathologists labor under difficulties, because they get the eyes in the last stage of the disease. They cannot examine the condition of the acute glaucoma. They see the eye afterwards; they see the result of secondary processes. That is the excuse the pathologist offers for not furnishing us better light on the subject.

I certainly cannot take any other ground than that Dr. Fiske has proposed, to perform an iridectomy as early as possible after the glaucoma has set in. I also endorse his position in regard to performing an iridectomy on the second eye, if the eye shows any premonitory symptoms. I would, however, not perform an iridectomy on the other eye if it shows a perfectly normal condition. It might not become glaucomatous for ten years or longer.

DR. SUKER.—In speaking of the pathology of glaucoma, I think it might be advisedly considered a uveitis of some kind. I agree with the doctor that we should do an iridectomy in both eyes, in one as a preventive when the other eye is attacked with glaucoma.

I would not use 2 per cent solution of eserine; even 1 per cent is irritating at times. Although it is used, I think it is not good practice.

I do not believe an iridectomy will do much good when you have a deep anterior chamber.

DR. ALT.—I wish to say to Dr. Suker that, pathologi-

cally, glaucoma is not found to be uveitis. From the examination of a very large number of glaucomatous eyes I am firm in the conviction that this is not the case, although a secondary glaucoma may ensue in cases of uveitis.

DR. FISKE (closing discussion).—My reason for writing the paper was to make a plea for the early iridectomy, and I was pleased to hear most of the members who spoke agree with me. In Zürich, Prof. Horner first advised me to do iridectomy for simple glaucoma.

I do not agree with Dr. Suker about the uveitis. With 2 per cent eserine I have never had irritation.

I have never seen a case of glaucoma where there was distinctly a deeper anterior chamber than in the ordinary eye where there were no adhesions between the iris and the lens. I do not think you have normal vision in those eyes where there have been attacks of glaucoma.

HOW TO AVOID SECONDARY OPERATIONS AFTER CATARACT EXTRACTION.

By C. BARCK, M.D.

ST. LOUIS.

THE endeavors, in cataract, to reestablish useful vision by one operative procedure are old. Unhappily the ideal method—removal of the lens in the capsule—as aimed at by Pagenstecher and others, can, up to the present time, be safely accomplished by no proposed technique.

Some years ago, the cystotome was replaced by the capsule forceps, which is still used by many operators. Others, among them myself, have again discarded the latter, partly because the results were not much more certain than with the former and partly on account of some dangers connected with its use. But one of the objects in selecting this theme was to hear the views of those present on this instrument and to find out how much it is in use to-day and with what success.

The oldest method of dealing with the capsule was a crucial incision in the pupillary area, which often opened it imperfectly, so that capsular cataracts were frequent. Then Knapp introduced about 14 years ago his “peripheric horizontal incision” in the upper third of the capsule. Secondary operations became still more frequent. This was to be

expected since the larger part of the capsule, the lower two-thirds, remains intact *in situ*. Knapp, with his method, accepts the necessity of the secondary operation as the rule rather than the exception. In his second series of a hundred cases of simple extraction, he performed discission in 74 instances (*Archives of Ophth.*, 1889). The immediate result, as regards the vision, was not once equal to $\frac{20}{20}$; after discission it increased to $\frac{20}{20}$ in 30 cases. In his third hundred (*Archives of Ophth.*, 1890) he made a secondary operation 53 times. By the primary operation he secured a vision of $\frac{20}{20}$ in only one case; by the secondary this was obtained in 20 additional ones. But Knapp adds that he feels certain that many cases of this series would present themselves for secondary operation later on.

I had practised the peripheric horizontal incision for a number of years before I gave it up on account of the number of secondary operations which it necessitated. I need not dwell here on the inconveniences which they cause, especially in patients living at a distance, who must come a second time to the city. Furthermore, although it is small, still there is a certain amount of danger connected with discission. From my personal experience I recall one case of a glaucomatous attack following discission which, however, subsided without any permanent damage. Others report similar consequences; still others, acute or subacute irido-cyclitis, with its dire sequelæ.

About three years ago I abandoned this method, and have gradually developed the one which I now use. It is a return to the old method, with some modifications. I lay the main stress on a long, vertical incision, commencing inferiorly, going with the cystotome between the iris and the lens capsule downward to the very periphery of the lens and dividing by an upward movement the lens capsule as extensively as possible. At first I added to this a horizontal incision both right and left. In order to simplify the procedure and to reduce the strokes of the cystotome to two, I devised the following method. The first incision is of a crescentic shape, commencing laterally from the lower end of the vertical meridian; the second one commencing just as far mesially from this, meets the first somewhat above the center of the capsule. (See drawing). It is very important that the two incisions

really intersect. If correctly carried out, the lower triangular flap as a rule falls downward, and the upper portion retracting, leaves a clear, central pupillary area.



In a number of instances, the remnants of the capsule placed themselves so that they just filled out the coloboma left by the iridectomy. Such a result is of course beneficial both from a cosmetic as well as from an optical standpoint. In five of my last 50 cases I had to make a discission, that is in 10 per cent. In two of these the extraction had been followed by iritis causing a closure of the pupil. In the third, the patient had been dismissed with good vision; some months afterwards a severe infectious disease was followed by an iritis and, in consequence of want of proper treatment, by a closed pupil. The number of cases, therefore, where the obstruction was due to the capsule alone, is a very small one. After these very satisfactory experiences with this form of incision, I feel justified in recommending it to you for further trial.

DISCUSSION.

DR. ALT.—The advantage I can see in the incision of the capsule recommended by the essayist is that no upper capsular flap is formed which may become engaged in the corneal wound. This, however, can be avoided in other ways, especially by the peripheral capsulotomy, with vertical section added, as I always practice it. As ingenious as Dr. Barck's capsulotomy may be, I do not see how any form of incision in the anterior lens capsule can prevent a secondary cataract from forming and necessitating a secondary operation. It is not the anterior but the posterior capsule, in my experi-

ence, which by wrinkling and slight tissue-formation gives rise to what we term a secondary cataract. I fail, therefore, to see how this incision of the anterior capsule can be of value in preventing it. Nothing but the removal of the lens capsule can do so, and even then the formation of fine connective tissue threads may produce a quasi-secondary cataract. This, also, happens when, as Hasner recommended, the posterior lens capsule is incised as soon as the cataract has been expelled. I have often done this, and abandoned it, since it did not prevent the formation of a very thin membrane which obstructed the former opening and which undoubtedly resulted from the inherent injury to the anterior parts of the vitreous body. It seems to me that no method of extraction can insure against the necessity of a secondary operation, but surely no kind of incision in the anterior lens capsule can do so.

I should also think that Dr. Barck's incision leaving a pouch at the upper periphery of the capsule might interfere with an easy delivery.

DR. STILLSON.—I am in the habit of trying to make a round incision. My chief difficulty in my last fifty cases has been from a little membrane or veil which forms after the extraction of the cataract. This I take to be the remains of the posterior capsule of the lens or the hyaloid membrane which marks the anterior boundary of the vitreous. If the doctor will tell us how to get rid of this interference to vision I will take it as an especial favor. The opacity of the anterior capsule after an extraction is certainly an undesirable outcome to what we so often know to be a really good extraction; and I never liked the idea of leaving this for a secondary operation, so I have as a rule not only incised the capsule freely, but I always wash out the pieces and frequently go in after the capsule with forceps; but as before mentioned it is the deeper membrane which bothers me and which I never like to pierce with a knife if there is any way to avoid it.

DR. FISHER, Chicago.—Dr. Barck's paper is certainly very interesting, and anything that will add to that contribution will be very valuable. In a paper of this kind, that is of so much importance, I think that Dr. Barck should give us more details regarding the vision after operating. I am of

the opinion that his paper would be more valuable if he would group his cases and give us his results.

We do not know what vision Dr. Barck is satisfied with after a cataract operation. The operation is an ingenious one, and if Dr. Barck gets good vision in all his cases without a secondary operation, it is certainly a very valuable one. If we are satisfied with $\frac{20}{30}$ or $\frac{20}{40}$ or $\frac{20}{50}$ of vision, we might in many cases avoid operation. I would like to ask Dr. Barck what he regards as sufficient vision to avoid secondary operation.

DR. HOTZ.—The only way to avoid secondary cataracts is to remove the lens within its capsule, no matter what ingenious incision you may make. It is not the anterior capsule which gives the most trouble. We find afterwards a fine veil spread over the pupil, and that is the posterior capsule, dusted over with fine deposits, as the result of some slight uveitis or hyalitis following the operation. No ingenious splitting of the capsule will avoid secondary operations.

DR. ROGERS.—This subject is very near to my heart on account of the attention given it by my associate, Dr. Clark, and I have made a rough diagram on the board which will illustrate a method devised by him about eight years ago and since then used by both of us when practicable. Two vertical incisions are made in the capsule on either side of the pupillary space, extending well towards the periphery; these are united by a cut that crosses their upper limit, extending three or four mm. beyond them, if possible, parallel with the periphery of the lens. In this way a flap is formed as Dr. Barck has illustrated, without the disadvantage of the lens occasionally rotating. Occasionally the little flap can be cut across below without undue traction or instrumentation, in which case the result is most satisfactory. I agree with Drs. Alt and Hotz that it is absolutely impossible to get a perfectly clear opening entirely free from even a diaphanous sheet unless the lens is removed in its capsule. But certainly the nearer we can come to it the better.

DR. GREENE, Dayton, O.—The ideal operation for the extraction of cataract, when we wish to eliminate all chance of a secondary operation, is to extract the lens within the capsule. The next method of operating, which at all ap-

proaches it in efficiency, is that of extracting the anterior leaf of the capsule. By this method we accomplish two things. We make the capsulotomy and avoid the secondary wrinkling and opacification of this portion of the capsule leaf so commonly seen a year or more after the extraction has been made. One or the other of these methods should always be employed in hypermature cataract, in operable cases of complicated and in all cases of traumatic cataracts where the capsule is thickened from inflammatory changes. For some time past, with the idea in view of so opening the capsule that it would by virtue of its elasticity withdraw from the pupillary area, and thus avoid, when possible, the necessity for a secondary operation, I have been opening the capsule by crossed incisions, using two cystotomes, one with its cutting edge parallel with the shank of the instrument, this to cut from below upward; the other with its cutting edge at a right angle with the shank, with which the lateral cuts are made. The result is a division of the anterior leaf into four sections, which have gotten out of the way in a very satisfactory manner in the limited number of cases in which I have followed the method. Next year I hope to have more to say about this method and the visual results obtained.

DR. BARCK (closing discussion).—In regard to what Dr. Alt has said, will say that I have never seen any difficulty in expressing the lens. On the contrary its delivery, with this section, is a very easy one. Furthermore, since I perform such an extensive laceration of the capsule, I do not recollect seeing a single prolapse of the vitreous.

As to the shriveling or shrinking of the posterior capsule, which becomes apparent only years after the extraction, this cannot be prevented, of course, by any form of capsular incision, but only by the extraction in the capsule. However, my paper plainly stated that it did not deal with these, but with the immediate secondary cataracts due to the presence of the anterior capsule within the pupillary area.

As a rule I am satisfied with a vision $\frac{20}{30}$, and would try to improve this degree by discission only under exceptional circumstances. In the instances where the vision was a lower one this was not due to the presence of the anterior capsule within the pupil.

ELECTRO-CAUTERY TREATMENT OF CORNEAL WOUNDS AND ULCERS.

By JNO. A. DONOVAN, M. D.,

BUTTE, MONTANA.

FROM the natural conditions of the city in which I live, mining and treating ores is the principal industry. In such occupations, corneal injuries are of the most frequent occurrence. In my ophthalmological work, conditions resulting from corneal injuries constitute a great portion of my practice; in fact, much more than any other disease.

With the laboring man, time is an essential feature in surgical treatment, as everyone desires treatment by the method with which can be obtained the best results in the least possible time.

Until within the last three years I treated all wounds and ulcers by cleansing and endeavoring to maintain asepsis, and stimulating whenever necessary. I faithfully used iodoform, later zeroform and nosophine. For local applications, when indicated, I curetted, used 1 per cent. sol. of formalin, pure phenol or tinct. iodin. With these, in most cases, satisfactory results were obtained; in others the reverse was true. In some obstinate cases, I secured nice results from the use of cassaripe in a 10 per cent. ointment, on which I reported in *Oph. Rec.*, Nov. '99. However, when all else had failed and not till then, I would resort to the use of the electro-cautery, which method of procedure I am led to believe after a visit to many of our Amer. Oph. hospitals, is still the common practice, i. e. using the cautery as a last resort. I asked myself the question: if these obstinate ulcers heal so nicely after using electro-cautery as the last resort, why not use it at first? The frequency in the use of the electro-cautery in my rhinological work has changed, with increased experience, in inverse proportion to its use in ophthalmological work.

The average simple non-infected corneal ulcer or wound will heal just as readily if it is simply protected, kept clean and let alone, as it would with the most energetic treatment. Therefore, the practice of many surgeons of touching all corneal abrasions with iodin or phenol or any stimulant is, to say

the least, superfluous, not to mention the pain and reaction that always follows. As to the physiological explanation of the results obtained by use of the electro-cautery, there might be much said, but whatever its method of action, experience has taught its use in a great many cases is essential to get best results.

Most of my accident patients come from the mines or smelters with a piece of quartz in the cornea. There is no means of judging whether the speck is simply rock or contains irritating compounds of copper, arsenic, etc.; in others slight abrasions have already become serious by contamination with copper water, which is a concentrated solution of sulphate of copper that saps through the rock above and dripping down on the miner's head gets into his eyes. If the wound be but slight, I either give nothing or a boric acid and zinc wash, instructing the patient to return in 24 hours; if there then be any irritation, I lightly touch the spot with the cautery at a very dull heat. This is usually all the treatment required.

For nearly three years past, in all corneal ulcers, no matter what the origin, if they appear severe enough to require any further treatment than a mild cleansing lotion, I invariably, after putting in a few drops of holocain, clean out any excessive amount of necrotic tissue with curette and use the electro-cautery. The results have been more satisfactory, considering time, reaction and suffering, than I obtained with any other method of treatment. I use a current transformer on the light current and a short straight cautery point, placed in the handle at angle of 45° . The point is heated to a dull red in most cases, though in some I do not reach even the degree of red heat. In the ordinary cases, the lightest possible punctures are made all around the edge of the ulcer just in the edge of the healthy cornea, punctures being about $1\frac{1}{2}$ mm. to 3 mm. apart; also, when necessary, I touch any part in its floor that appears unhealthy. This latter procedure greatly hastens the healing process if there is any necrotic or unhealthy spots in the floor, but if it appears clean, this should be omitted. With few exceptions, this is all the active treatment necessary. If after a few days (say two to four) some places appear to have made no progress, these spots

should be re-touched. In the rare cases in which the ulcer does progress in some direction, the advancing portion should again be touched by puncturing just in the edge of the normal tissue; this acts apparently in the same manner by which prairie fires are controlled by burning an area in front of them.

I claim no originality as to the method of corneal punctures at the edge of ulcers. I first received the suggestion of it from Dr. Herman Knapp. He later demonstrated the method before the Oph. Sec. of N. Y. Academy of Medicine, Nov. 1900, as reported in Vol. xxx, Archives of Ophthal. I quote a paragraph from a letter received from Dr. Knapp under date of New York, March 9, 1903. He says: "I have tried this method ever since, and used it almost to the exclusion of any other procedure. It is only lack of time kept me from publishing the results of the method, which are in general very satisfactory, frequently surprisingly good. I have dozens of carefully recorded cases in my record books and shall publish them as soon as I can make time for it."

With children, I formerly gave chloroform, but now use only local anæsthesia. Spots in the conjunctiva require more holocain than in the cornea. Cauterization can be accomplished perfectly by handling the cautery with the same precaution we use the cataract knife, and touching each spot instantaneously. A speculum or a fixation forceps is usually not necessary. With this treatment the child requires nothing more.

In wounds of the cornea, when it has been completely perforated, cut or lacerated, and probably not aseptic, I touch the entire margin of both edges of the wound. If in apposition, this coagulates any exudate and forms a protective covering; it at the same time cements the edges together. To illustrate: Patient aged 65; prospector; struck in a saloon with a broken beer bottle at midnight. Consulted me at four P. M. next day; found vertical cut extending from 1 mm. above to 1 mm. below sclero-corneal margin, including the entire center of the cornea; protrusion of the iris above and below. I cut off both protruding portions of the iris with scissors, then cauterized full length wound thoroughly; this practically sealed it. Patient left next day for his cabin in the hills,

taking with him only atropine solution. Saw him next in six months' time; had no more pain; made nice recovery and had useful amount of vision.

To illustrate results obtained in severe ulcerations: Mrs. B. aged 63; wash-woman; ulcer covering a third of cornea; hypopyon extending up to pupillary edge. At first visit in my office, I used cautery. She walked home and returned third day; pus had disappeared; no pain; simply kept eye covered. She made an uninterrupted recovery.

On the evening of the same, Mr. P. aged 55, mining engineer, consulted me; was injured two weeks previously. The local condition was nearly identical with that of Mrs. B. Used cautery; extension towards the center of the cornea stopped but extended toward the periphery. Three days later I touched the advancing edge and opened the anterior chamber. I repeated this three times within nine days. Then he entered the Murray & Freund Hospital and was put to bed; second day pus was gone and he resumed work three weeks later.

Girl aged 2 yrs.; corneal ulcer; treated with phenol once; later iodine twice; with continued use of drops, and nosophene ointment, fully recovered after two months. When ulcer suddenly recurred, I applied pure phenol but did not check its progress; on the third day, under chloroform, used electro-cautery. No more active treatment, and now after three years, there is but the faintest nebula, not perceptible to her parents. This being the first time I used it on a child, and several almost similar cases following, induced me to substitute local anæsthesia for chloroform, and use the cautery as primary treatment. The time consumed from making a diagnosis till the patient is operated on and leaves the office does not exceed 10 or 15 minutes.

To illustrate its use in apparently simple cases: A child with a mere abrasion caused by a finger nail, was brought to my office on account of pain. I prescribed holocain, but on third day being informed the mother was compelled to use it several times each night, I touched the spot with cautery point barely at the temperature where it burns cotton (this is the test I use to regulate a black heat). After three hours, pain was gone and no further treatment was used.

CONCLUSIONS:—Whenever a corneal wound or ulcer is se-

vere enough to require treatment, electro-cautery is indicated. With proper appliances in careful, competent hands, its effects are absolutely controlled and is perfectly safe with patients of any age. The results are better, quicker, more certain, with less reaction and much less pain than can be obtained by the use of phenol, iodine, formalin or other strong stimulant. The scar resulting is no more and frequently much less than would have resulted had any of the so-called less radical means been employed. I have used it in a great number of cases; so far I have penetrated through the cornea but twice, and then without any bad results, and I consider it in every respect one of the most satisfactory operations performed in ophthalmic surgery.

DISCUSSION.

DR. GAMBLE.—I think this is a very interesting paper. Dr. Donovan speaks of a solution of zinc and boric acid in treating these corneal ulcers. I would, myself, hesitate to use zinc in the treatment of a corneal ulcer. My experience is that we cannot use irritating substances, and our antiseptics are all more or less irritating. Zinc in my hands is a source of danger. I was glad to hear the essayist speak about the avoidance of scars. I have not been so fortunate. I think there is more danger with the actual cautery than with carbolic acid or nitric acid of producing scars. The action of these solutions is hindered by reaction of the tissues. With the proper use of carbolic acid or nitric acid, there are few ulcers we cannot control. It has been my experience that when the cornea is perforated it will take care of itself. I never have had occasion to cauterize the margins of wounds after perforation or incised perforating wounds. It might be that the infections from the mines would necessitate such treatment.

DR. SUKER.—The doctor is to be complimented on the excellence of his paper. He speaks of zinc sulphate and boric acid; they are chemically incompatible. I would ask if he has tried the use of nitric acid. It is a stimulant and a cautery at the same time. You can limit its action as nicely as you can the electro-cautery. I think the conclusions he draws are good. If the electro-cautery, as a last resort in some

cases does good, it would accomplish the same as the "first resort."

DR. BRADFELD.—I do not believe zinc sulph. should ever be used in corneal lesions, as it is very irritating, and when an astringent is needed one should be chosen over which we have better control. In superficial infections of the cornea I very much prefer fuming nitric acid to the actual cautery, as it is much less liable to leave opacities.

DR. CONKEY, West Superior, Wis.—While the electric cautery is an excellent remedy it has its limitations. It will not always stop the progress of rapidly spreading septic ulcers. I have lost some eyes by depending entirely upon it. In these malignantly destructive cases it should be combined with free incisions through the ulcer. The anterior chamber should be opened and kept open till the ulcer begins to heal. The fluid from the anterior chamber seems to exert a some powerful antiseptic action upon the diseased surface than does the cautery.

DR. G. F. KEIPER, Lafayette, Ind.—I believe that taking all in all, the galvanic cautery is the best means we have at present for treating corneal ulcers. I recently cauterized an eye and re-cauterized it, and the ulcer continued to spread, and I then used nitric acid and finally iodine. I also recall a case where I was obliged to cover the place with normal conjunctiva in order to prevent the escape of aqueous according to the method of Kuhnt. As a rule a burn in the cornea is easier to heal than an ulcer.

DR. M. D. STEVENSON, Akron, O.—It is most important in using the cautery to apply it only momentarily and then remove it, reapplying it as often as necessary, much depending on the efficiency of the apparatus. The aqueous will become heated and cataract result, if it is held too long in contact with the cornea. When the base of the ulcer is thin, often presenting a slight bulge as if it is going to rupture, it is well to perforate it with the cautery, thus reducing the tension of the eye, and keep the opening patent until the eye is much improved. I always instill a weak solution of fluorescein into the eye to more clearly outline the ulcerated area. In ordinary round non-progressive ulcers I cauterize the area of first staining, but in one that is rapidly progressive in a

certain direction I also cauterize the area of second staining in the same direction, which extends 1 mm. or slightly more into the adjoining infiltrated cornea. A nervous patient should not be informed of its use, as through fear their co-operation may be lost and damage result. I have used nitric and carbolic acids, properly applied so that there was no excess to run over the cornea, much more frequently than the electro-cautery and consider that in ordinary cases they are as good and more easily and safely applied. The electro-cautery is not necessarily the best to use in all cases merely because it often is more useful in some of the severer types.

DR. JOS. TITCOMB, Duluth, Minn.—I believe cautery to be the best single agent we have. Personally I prefer the actual cautery by means of the platinum probe heated at the alcohol lamp. The cautery is the agent that does the work, and unless the ulcer is central, where there is possible danger of opacity, I almost invariably use it.

DR. HECKEL — I do not think any one doubts the efficacy of the electro-cautery treatment. I like to use carbolic acid in small ulcers. It gives very good results. In certain cases nothing will take the place of the cautery. This is especially true of the class of cases Dr. Donovan describes, where it is excellent treatment. The use of some germicidal agent in conjunction with the electro-cautery enhances its efficacy. I formerly used silver nitrate with good results, but now use protargol in solutions of 10 grs. to 20 grs. to the ounce. It is a good germicide and has a beneficial influence.

DR. J. W. SCALES, Pine Bluff, Ark.—I find that locality has a great deal to do with the result of treating corneal ulcers. I dare say that if the usual treatment was carried out in my part of the country that is usual in the hospitals of New York and other places, the majority of the eyes would be lost. I am speaking of the urgent case that comes to the specialist in that locality. In the first place, we have a man with a large spleen and cathectic diathesis, his liver inactive and in a general asthenic condition. Any sort of stimulation will be detrimental to that patient. You first have to rouse his secretion, and the best thing in Arkansas is calomel. I would not hesitate to give my patient, if the occasion were urgent, at least 20 to 25 grains of calomel in order to get an

immediate effect, that is, within a few hours. The average case would not, of course, take as large a dose. Until you get the slight reaction, which will be indicated by an increase in his appetite, which is the best indication we have of a stimulation in the liver, we are compelled to use palliative remedies. The best I think is hot applications. If we use the cautery we will get one result in one case and another in another. My patients do not pay enough attention to the diathesis.

DR. WILDER.—I concur with the propositions of Dr. Donovan in the main. I cannot agree to his ultimate conclusion that because this treatment is good as a last resort, it is good as the first in every case. I do not believe that we should use this rather severe treatment in such a simple thing as a scratch of the cornea from a child's finger. In the hands even of a skillful person, the cautery will make quite a scar, and wherever you burn the cornea you will have a scar; if that happens to be central it may interfere with vision. I do not believe we should resort to this before we have tried antiseptic irrigations, etc. I do agree with him on the value of the treatment, and I rely less and less on carbolic acid and nitrate of silver in these cases. One practical point is that when the corneal ulcer has extended deeply, there will be a little knuckle of the membrane of Descemet sticking up like a little pearl. One may cauterize the advancing border, but the keratocele will remain and prevent the healing of the wound. Not until a puncture is made through the keratocele will the process stop. As Dr. Stevenson has said, we relieve the tension in these cases and accomplish just what is done by the Saemisch incision.

DR. DONOVAN (closing discussion).—I use zinc, one to two grains to the ounce only, as a rule, and in that proportion it is hardly a stimulant; it is but a mild astringent. It has been said that the perforating wounds usually take care of themselves. It is true, a great many of them do. I do not mean to say that you should use the cautery when the cornea seems to be doing all right. But if you know it is infected, it is not well to wait to have it demonstrated. I did use nitric acid on several occasions, and I have forgotten just what my results were. I use the electric cautery so fre-

quently that I think I can do better than with nitric acid. It is not necessary to cauterize to the bottom to stop the infection. As a rule, I make the slightest possible punctures. I cannot get these patients to go to bed. I think every one with a sore eye should go to bed, but I cannot get them there. In spreading ulcers it checks them as a rule. I just touch the ulcer with the cautery as lightly as possible — just touch it and withdraw. I do not use fluoresceine any more. As a rule the patient has diagnosed the trouble and outlined the treatment, and if the ulcer is extensive enough to need treatment, it shows. I consider the electro-cautery safe, but not to be used too freely in the center of the cornea. Boracic acid and zinc sulphate I do not consider incompatible. It makes an absolutely clear solution and you get an astringent effect from it.

TUBERCULOSIS OF THE IRIS, WITH PRESENTATION OF MICROSCOPIC SPECIMENS.

By WM. H. WILDER, M. D.,

CHICAGO.

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SOME difference of opinion seems to exist as to the frequency of this disease. Wagner (*Münch. Med. Woch.*, 1891, Nos. 15 and 16) states that 50 per cent. of all cases of iritis are of this character. Michel (*Lehrbuch der Augenheilkunde*) also regards it as very common, and states that forty or fifty of every hundred cases of iritis are tuberculous. Both authors seem to include in their category all cases of serous iritis in which there are small masses of exudate in or upon the iris, that simulate in any manner tuberculous nodules. Horner estimates that it will not be observed more frequently than once in four thousand cases of eye diseases, while Hirschberg, of Berlin, saw only six cases of it among sixty thousand eye cases.

Velhagen (*Klin. Monatsbl. f. Augenh.*, XXXII p. 121) says that among eight thousand patients in the Eye Clinic at

Göttingen, no case of tuberculous iritis was seen. With these latter opinions I think most ophthalmologists will agree, and we must conclude, therefore, that it is a rare affection. Probably early observers who had opportunity for examining such cases, considered them as either condylomata or sarcomata, or placed them in the category of idiopathic iritis; but with the invention of the ophthalmoscope and the discovery that tubercles develop in the choroid in connection with either general or meningeal tuberculosis, a more careful study of certain inflammations of the iris and ciliary body has determined their exact nature. Cohnheim demonstrated that tuberculous iritis can be caused by introducing particles from tuberculous lymphatic glands, granulations of tuberculous joints, etc., into the anterior chamber of the eye. More recently investigators have caused the disease experimentally by injecting pure cultures of the tubercle bacilli into the aqueous chamber. The introduction of small masses of suspected tissue into the anterior chamber of the eye of a rabbit is employed as a means of determining whether such tissue is tuberculous. About twenty or thirty days after the injection into the eye, small, reddish grey nodules make their appearance in the iris and the eye becomes inflamed. The nodules increase in number, coalesce and fill the anterior chamber with a mass of new tissue. The cornea becomes involved and the growth breaks through, thus destroying the eye. The animal usually dies from general tuberculosis.

Tuberculosis of the iris manifests itself in three forms:

First, Solitary Tubercle.

Second, Disseminated Tubercle.

Third, Simple, inflammatory tuberculous iritis (Michel).

The solitary growth affects one eye alone and develops as a small, round or oval nodule, usually at the periphery of the lower part of the iris. It is grayish red in color, and as it grows it fills the anterior chamber of the eye, and much resembles a neoplasm. Indeed such a mass may easily be taken for a sarcoma. This also goes by the name of granuloma, a term, according to Fuchs, that was given by von Graefe, "because Virchow who made the anatomical examination of the tumor designated it as granulation tissue." This term should be abandoned, for it is misleading and liable to create

confusion. As the growth increases, the cornea becomes involved and perforates, allowing the mass to break through, which presents as a pale yellowish or grayish mass similar in appearance to granulation tissue. It does not then continue to grow, but breaks down, and the eyeball gradually begins to shrink as the inflammatory process occasioned by the growth subsides. The eye is, of course, lost and phthisis bulbi ensues. General tuberculosis infection may result.

In the disseminated form which may occur in one or in both eyes, there are at first all the symptoms and signs of an iritis. Soon little yellowish grey nodules surrounded by a slightly reddened zone, appear in the iris. These vary in size according to the growth, but are from one to six mm. in diameter. Their favorite site is at the root or periphery of the iris, and they seem to be constantly changing, some disappearing while others are forming. Their predilection for the outer or root zone of the iris helps to distinguish them from the condylomatous nodules, so frequent in syphilitic iritis that usually are seen at the pupillary margin. Graefe describes them as of the size of millet seeds, distributed over the iris, especially over its lower half and some distance from the pupillary margin on the *circulus arteriosus minor*. Some of these tubercles may disappear completely, leaving small patches of atrophied iris, while others may coalesce, forming larger tuberculous masses that fill-in the angle of the anterior chamber.

The iris is dull and discolored, and flakes of lymph and disintegrated tuberculous nodules may fill-in and occlude the pupil. Firm and extensive adhesions form between the iris and the lens, and the tension of the eye may become markedly increased. Lubrowski (*Archives of Ophthalmology*, Vol. XXIX, No. 3) reports several cases in which glaucoma supervened. Ciliary injection is marked and the eye is very often sentive to pressure. The ciliary body and choroid may be invaded, and even the cornea may be involved, the tubercles presenting the same general appearance in that structure as in the iris.

As the case progresses, there may be bulging of the ciliary region and even perforation; or the process may subside with gradual shrinking and atrophy of the eyeball. Tuberc-

cular meningitis or general tuberculosis may supervene, so that the prognosis, both general and local, is bad.

In the third form, according to Michel, the tubercles are not clinically demonstrable, being situated in the tissue of the iris and not on the surface. The disease assumes the form of a chronic iritis or irido-cyclitis, which causes either complete annular posterior synechia of the iris, or adhesion of its posterior surface to the lens. In the former, there would be iris bombée and more or less atrophy of the tissue of the iris. In the latter form there is proliferating inflammation of the iris which becomes hypertrophied, and granulation tissue fills the posterior chamber. The tuberculous nature of the trouble may be demonstrated by excising a piece of the iris and examining it microscopically for the existence of tubercle. Deposits of lime are frequently found in such irides, and even true bone formation has been observed as in the choroid.

Some observers, Leber (*Bericht der XXI. Versammlung d. Ophthalmol. Gesellsch.*, Heidelberg, 1891), Samelsohn (*Bericht der XXIII. Versammlung der Ophthalmolog. Gesellsch.*, Heidelberg, 1893), and Van Duyse (*Archiv. d' Ophthalmologie*, XII, p. 478), describe a form of attenuated tuberculous iritis, presenting all the salient features of the disseminated form, which may end in spontaneous recovery, the functions of the eye being partly or wholly preserved. This form is slower in its course and affects older persons.

All of the forms mentioned present the same histological features, differing in degree, namely the typical tubercle formation of round cells around a central giant cell. This giant cell is a large crescentic or round structure with non-granular protoplasm, containing near its periphery numerous elongated nuclei. The little tubercle mass is frequently seen on the wall of a vessel, and develops from the adventitia. This is one means of differentiating histologically a tubercular from a syphilitic node, for in the latter the growth begins in the intima and the lumen of the vessel is blocked. In the milder forms of the disease, the bacilli are very scarce and difficult to find. It is assumed, therefore, that in some of these cases the exciting cause is not so much the direct action of the bacilli as it is the irritation caused by the toxines generated by them, circulating through the delicate tissue of the

iris, which, for some reason, is unusually sensitive and susceptible.

This is a disease of childhood and adolescence, the large majority of such patients being under the age of twenty years. Of 121 cases reviewed by Schieck (*Graefe's Archiv. für Ophthalmologie*, Bd. 50, part 2, 1900) ninety-six were under the age of 20 years; while of the remaining 25, only six were over thirty years. The oldest was 55.

Most of the patients affected with tuberculous iritis have either pulmonary tuberculosis or tuberculous manifestations in other structures, such as joints, lymphatic glands or skin. Some have a bad family history, while a few have neither a family nor a personal history of tuberculosis, nor do they show any manifest signs of the disease, except in the lesion of the eye. Such cases raise the very interesting and important question, whether the iritis is primary or secondary to some other tuberculous lesion, and many capable observers take the view that it is primary. It is argued that the eye of a sound person may be infected locally through an abrasion of the cornea, or an ulcer of the cornea or conjunctiva, and that in such manner the bacilli may gain entrance to the deeper circulation of the eye.

DISCUSSION.

DR. ALT.—Tuberculosis of the iris is a comparatively rare disease, although possibly more frequent than we know. The statement that from 40 to 50 per cent. of iritis are tuberculous, seems to be based on the paper of Michel, published in Graefe's Archives some 18 or 20 years ago, in which he tried to show that in iritis the inflammation is always a nodular one. At a certain stage this is really the case in many forms of iritis, but while the picture is suggestive of tubercles, these are not tubercles. It is due to the accumulation of leucocytes around all or at least a large number of the very many blood-vessels in the iris. While the specimens shown by the essayist are very fine and characteristic I believe that in modern times the diagnosis of tuberculous iritis is often made without a warrant. Personally, I have not yet seen a case, although I have seen some specimens which were certainly tuberculous.

DR. SUKER.—I would like to ask whether in the so-called acute variety the arrangement of the tubercles and cells is

similar to that in chronic tuberculosis, and whether or not the coalescence in the iris is as marked as in the single tubercles of the choroid. My experience in tuberculosis of the iris has been mainly experimental. If any of you wish to try it, you can take a small syringe and through a small corneal incision inject a pure culture of the germ. In 34 hours or later you will see the characteristic growth begin and you can watch it nicely. Rabbits are especially susceptible.

DR. JOS. BECK, Chicago.—This case of Dr. Wilder interests me particularly from the histo-pathological point of view. I have been studying this specimen microscopically and have found some beautiful giant cells, characteristic of tuberculosis. I had the pleasure of seeing tubercular iritis and irido-choroiditis at Graefe's and Wintersteiner's laboratories. The differential diagnosis between syphilitic and tubercular iritis based on the presence of giant cells, when found alone, is not absolute, because one can find them in both the conditions. The endarteriitis obliterans in syphilis and the periarteriitis in tuberculosis are points of greater diagnostic value. Another point in the diagnosis is the reaction to tuberculin, which was not mentioned in the writer's paper, I believe. In Elschinig's case of tubercular irido-choroiditis the experiments with tuberculin were carried out and a reaction obtained.

DR. M. D. STEVENSON, Akron, O.—It is clinically important but often difficult to differentiate between tubercular nodules, condylomata and sarcomata. The former usually occur in young people with a tubercular history and their size, peculiar grayish white or yellowish gray color, and lack of bloodvessels help to distinguish them. The condylomata occurring in older people, with a history or signs of lues are usually quite small and vascular and quickly disappear under specific treatment. Non-pigmented sarcoma in old people is always single and very vascular, it steadily increases in size and never disappears. Microscopically this can be easily differentiated although all of the tumors may have giant cells. It is important to note whether the inner or outer coats of the vessels are chiefly affected.

DR. WILDER (closing).—In reference to the point raised as to the giant cell and its differentiation from the giant cell of syphilis, one must bear in mind there are several varieties

of new growth in which the giant cells may be found. They are found also in sarcoma and may make one think of tuberculosis. But there is a way of telling them apart: the giant cell of tuberculosis is usually circular in outline or elliptical, rather symmetrical, while that of sarcoma is more angular, and irregular. It is the same in syphilitic forms. Another point is that the contents of the giant cell of tuberculosis are clear and non-granular, while those of sarcoma are granular. Also, the nuclei in the tubercular giant cell are peripheral and usually elongated, while the others are central. Another important differential histologic point is that in tuberculosis the growth begins in the adventitia, while in syphilis the process usually starts in the intima of the vessels. In regard to the reaction of tuberculin, I think it uncertain, at least it has been so in the limited experience I have had. I have had two cases in my own practice where there was no reaction after the use of tuberculin.

(Dr. Wilder showed a number of microscopical specimens).

MYDRIATICS IN REFRACTION OF PRESBYOPES.

BY O. A. GRIFFIN, M. D.

ANN ARBOR.

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SINCE the introduction of cycloplegics into refractive work, there has strangely existed, among ophthalmologists, a great diversity of opinion as to the necessity and advisability of employing these drugs as an aid in the determination of ametropic conditions. These contentions have been confined more particularly to the propriety of their use in the refraction of adults, while their employment in testing the presbyopic ametropes seems to be quite generally regarded as not only superfluous but attended with risk of inducing a glaucomatous condition. With a view of eliciting an animated discussion upon these points, I beg to present a few remarks upon the use of mydriatics in the refraction of presbyopic eyes.

When a patient presents himself for relief from an ametropic eye strain, be he young or old, the same difficulties of

making an accurate estimation of the refractive condition of the eye, without the aid of a mydriatic, obtain to a greater or less degree. The variability in refraction of an accomodating eye, the dependence of a subjective test upon the uncertainty of a patient's judgment, the frequent latency of hyperopic conditions, the fact that a sphere will improve to a certain extent the vision in astigmatism (although this phenomenon is denied by many writers upon refraction), the additional observation that 90 per cent. of my refractive cases are astigmatic, one-quarter of a diopter or more, and most important, an inability of properly employing the objective tests as a means of corroboration have convinced me, after making several thousand refractions, that the aid of a cycloplegiac is not only necessary to the accomplishment of an accurate refraction of the young adult, but may be frequently employed with profit in the testing of presbyopic ametropes. Frequently is the statement made that it is not necessary to correct the smaller degrees of astigmatism when prescribing for an ametropic defect in the presbyopic. Such reasoning seems to be pregnant with anything but logical thought. With as much propriety might a physician consider his duty performed after administering a quieting potion to relieve a chronic disorder. If it is necessary to correct an ametropia at all, why burden a patient with a glass that removes only a portion of his trouble, when every vestige of strain and discomfort might be eradicated thereby? It is frequently the presence or absence of these small defects that decides the comfort of the patient.

To briefly illustrate the validity of my position with reference to this subject, I will cite a few instances in which a satisfactory refraction was not obtained until a mydriatic was used, when both the subjective and objective methods of testing were employed, the findings of the latter being usually preferred.

CASE 1. Mrs. C. R. W., aet. 49, consulted me for the relief of marked refractive symptoms from which she had suffered for several years, although she had repeatedly been fitted with glasses by different oculists. Vision was normal, conjunctiva chronically injected, blepharitis marginalis present, and general health greatly impaired. When she called upon me, she was wearing +1.00 sph. before each eye.

Without a mydriatic she accepted O. D. +1.00 sph. +0.25 cyl. ax. 60°; O. S. +1.00 sph. +0.25 cyl. ax. 120°. Not obtaining complete comfort from the use of these lenses, the eyes were homatropinized, when +0.75 sph. +0.50 cyl. ax. 60° was selected for the right eye, and +0.88 sph. +0.37 cyl. ax. 120° for the left, which was worked out principally by means of the skiascope, as the subjective tests were uncertain. An additional +1.50 sph. was given her for reading, with instructions to wear the distance glasses constantly. Though several years have elapsed, this same ametropic correction is being worn with complete satisfaction, and attended with a marked improvement of her physical condition.

CASE 2. Mrs. R. C., aet. 66, was referred to me by her physician for refraction. She had been unable to obtain relief from constant headaches and blurring of print from which she had suffered for two years, although she had worn several different lenses. Vision in O. U., $\frac{20}{60}$, was improved to $\frac{20}{20}$ by a 2.25 sph., which she was wearing. A subjective test, without a mydriatic, was attended with uncertainty as to the exact amount and axes of the existing astigmatism. Under homatropin, both of these points were decided without any difficulty, when she accepted in O. D. +2.00 sph. +0.75 cyl. ax. 180°, and in O. S. +2.00 sph. +0.50 cyl. ax. 180°, which was corroborated by the shadow test, giving a vision of $\frac{20}{15}$ in each eye. A +3.00 sph. was added in a pair of grab fronts for her presbyopic condition. Complete relief from her symptoms has been enjoyed since wearing these corrections during the past two years.

CASE 3. J. F. S., aet. 60, since a young man has had poor distant vision, pain in eyes, and blurring on reading, from which he has never been relieved, although he has often been fitted by several refractionists. Vision was $\frac{9}{200}$ in O. D., and $\frac{8}{200}$ in O. S. Was wearing -6.00 sph. for distance and -4.00 for reading. Subjective tests gave -6.00 sph. -1.50 cyl. ax. 115° for the right eye, while -7.00 sph. -1.50 cyl. ax. 60° was selected for the left, giving $\frac{20}{60}$ in O. U. A deduction of 3.00 diopters was given him for reading. After a trial of these lenses, which was not satisfactory, homatropin was instilled, when objective tests showed that the proper correction for the right eye was -6.00 sph. -2.50 cyl. ax. 120°, and -7.00 sph. -2.00 cyl. ax. 60° for the left,

which gave a vision of $^{20}/_{40}$. Together with an appropriate correction for near work, these corrections have produced a cessation of all his refractive symptoms since wearing them.

If space permitted, these typical cases might be augmented by the citation of a large number of similar instances, which have come under my observation and care. The difficulties of making subjective estimations as previously indicated are all illustrated by the above cases, and as the presbyopes, whom I usually meet, are from a distance and have generally consulted refractionists of more or less ability, if the usual methods of testing show any uncertainty in results, I do not hesitate to place them under the influence of homatropin, when a certainty of refraction is apparent. In following this procedure, I am actuated by a desire to please my patients in the removal of their distressing symptoms, and thereby further my reputation.

In regard to the risk incurred by the employment of homatropin in presbyopic eyes, it is scarcely necessary to add that, in my opinion, this matter has been greatly exaggerated by many writers, although as a routine, I employ a myotic to neutralize the effect of the mydriatic. Among the thousands of presbyopes, who, in clinical and private practice, have had *atropin* instilled into their eyes for weeks and even months at a time in the treatment of corneal and iritic disorders, how many have terminated in a glaucomatous condition? We employ mydriatics in these conditions with scarcely a thought of danger, but when suggested as an aid in the refraction of presbyopic ametropes, what a potency for harm it suddenly assumes in the minds of some! A reference to the literature of glaucoma reveals the citations of many instances resulting from the use of mydriatics, while a further search will show that cases have followed the instillation of eserine. Such contradictory evidence to my mind points to the purely incidental development of the glaucomatous state. Our present knowledge of the etiology, pathology, and in many respects even the treatment is quite uncertain as will be found upon investigation of the writings at hand, and until our knowledge upon these cardinal points becomes more definite and certain, can these harmful properties be justly attributed to the employment of mydriatics? I have yet to meet an instance in which the supposed increase of tension, or any other deleter-

ious condition has resulted from my use of the drug, either as a mydriatic or cycloplegiac. If the agent is employed as I have previously indicated, followed by a myotic, I am confident that our results in the refraction of presbyopic ametropes would prove more satisfactory, and the risk incurred practically nil.

DISCUSSION.

DR. SUKER.—This question of mydriatics is very interesting. I am glad to know that the doctor has become so expert that he can recognize with the retinoscope the difference between $\frac{1}{4}$ and $\frac{1}{2}$ diopter of astigmatism.

In regard to using atropine, as I have already stated, the ciliary muscle is constantly active; it does not undergo the sclerosis the lens undergoes. Therefore, the ciliary muscle, though still active, can not change the convexity of the lens. Beyond 65 years of age we know that atropine has no effect upon the manifest accommodation. The lens does not expand in its anterior-posterior diameter, though the ciliary muscle may or may not be paralyzed. Hence, if you get a complete paralysis, the refractive condition is not changed and the range of accommodation is not changed; therefore there is no reason for using atropine in these cases. Beyond the age of 45 I see no legitimate reason for the use of atropine in determining any kind of refractive error, excepting a temporary mydriasis for retinoscopic purposes.

DR. BRADFIELD.—I have no fault to find with the thorough examinations in refraction. It may rarely be necessary to use a cycloplegic after fifty years of age, but when done I insist that the proper glasses must be selected after the cyclopegia has entirely disappeared.

All the result produced by correcting low degrees of astigmatism in the presbyope, wearing plus 2.00 or stronger spheres, is purely in the mind of the operator, as a slight tilting of the glasses will much more than counteract the effect.

DR. MINOR.—I am very glad to hear the paper, for the doctor finally come to my way of thinking, after all. He did not agree with me last year when I said that I invariably used homatropin in every case of refraction, no difference about the age of the patient. I have yet the first time to find an increase of tension produced by it. I have read about it,

but although I have looked for it, I have never found it. The doctor told me that very often, if not always, he uses a drop or two of eserine in these presbyopes after the homatropin. This I do not find necessary. As for correcting astigmatic defects, I think this is highly necessary, and that is where the careful refractionist gets the good results in his work. I have cases every week refracted by other men who disregard this astigmatism, and I find that if I correct $\frac{1}{2}$ or $\frac{1}{4}$ diopter of astigmatism that the glasses are satisfactory to the patient.

DR. GAMBLE.—I think that the conclusions of Dr. Griffin's paper should be emphasized, and I believe thoroughly in every conclusion he has reached. I am not speaking from a theoretical point of view but from a clinical one. It has been my custom for two years to put homatropin in every presbyopic eye that comes to my office to be fitted. If there is any evidence of astigmatism, unless there is some contraindication. I think the use of homatropin is especially valuable in determining the required lenses in presbyopic cases for the purpose of revealing the astigmatism. I never feel sure the patient has a perfectly fitting lens until I have used the retinoscope. It is difficult to get a subjective test revealing the amount and axis of astigmatism, which is accurate. It differs with the personal equation of each patient. I do not see any objection to using the objective method, and you cannot get this without a cycloplegic. When through, you have the best possible result that can be attained.

DR. M. D. STEVENSON, Akron, O.—Always feels greater confidence in his result after the use of homatropin in presbyopes, and especially when he also gives a post-cycloplegic examination. He does not use it in all cases, and never when contraindicated by increased eyeball tension. His method is to instill ten or twelve drops of a 1 per cent solution in the eyes every five minutes (the patient waiting in a quite dark room), and commence the examination in from fifteen to thirty minutes after the instillation of the last drop. No trouble has so far resulted from their use, although eserine has occasionally been used after the examination as a preventive. Most all presbyopes have some accommodative power, and, if hyperopes, will usually demand too weak a lens. In photostcopy which the writer considers much the best objective method of examination, the refractive strengths of these

eyes without the use of a cycloplegic, are often noticed to vary much depending on their accommodation.

DR. GRIFFIN (closing discussion).—Dr. Suker compliments me upon my ability to diagnose between $\frac{1}{4}$ and $\frac{1}{8}$ diopter with the skiascope. I do not consider this as anything extraordinary, although I must say that these results are not obtained by a careless use of the method. Accuracy in this mode of testing is a matter of personal equation as in other methods of refraction. With the eye under a cycloplegic, I first use the subjective tests, and then the shadow method. In a comparison of results, with few exceptions, the patient accepts my findings of skiascopy as the best. This is not due to careless use of the subjective tests, but to the uncertainty of a patient's judgment. Normal vision through a lens, with or without a complete suspension of the accommodation, does not positively exclude a remaining ametropia. The doctor tells us that at 65 years of age the accommodation is suspended physiologically, but theory is one thing and facts are another. I have had patients 65 years of age where I could not make a reliable refraction without the use of a mydriatic; and even at 70, I have observed a variability in the tests. I recall a patient of 50 years, who complained of marked refraction symptoms, but so far as the subjective tests were concerned, no error was evident; though under homatropine, a compound hyperopic astigmatic condition of moderate degree was easily shown. Dr. Bradfield says that the patient does not accept the correction made under a mydriatic and the dilated pupil presents a different refraction than a normally contracted one. That is true in some instances of marked spherical aberration, but I overcome this defect by employing a perforated disc as previously indicated, whereby a central refraction corresponding to the normal pupil is made. Of course, that is done as a last test, when finally deciding between the subjective and objective findings. It is results that speak. It may sound all right to say that it is not necessary to correct $\frac{1}{4}$ to $\frac{1}{2}$ diopter of astigmatism in aged people; but when they come to the refractionist without this correction, and a remedying of these small defects results in a perfect cessation of the patient's former symptoms, which continues for years, I contend that it is not all imagination that produces these results. Again I say, results speak for themselves.

KERATOCONUS, ÆTIOLOGY, AND IMPORTANCE OF EARLY DIAGNOSIS AND TREATMENT.

By J. A. L. BRADFIELD,

LA CROSSE, WIS.

EARLY in my experience in special work my attention was very forcibly called to the meagerness of the articles in our text books on the subject of keratoconus and the paucity of the subject in our general medical literature. Time has convinced me that it is much more frequent and of more importance than generally considered.

I do not come before you with a large clinical experience all classified and tabulated, but will deal with the subject from my own experience; and if, after the reading of this, the paper is unhesitatingly criticized and the subject thoroughly discussed, the object of the writer will have been accomplished.

The paper will be limited to the typical keratoconus characterized by ecstasia of the cornea just below and to the inner side of the optical center. Keratoconus is a disease having its origin at puberty and characterized by asthenopia and gradual failing of vision both far and near. It occurs more frequently in the female, is usually binocular and often originates in the hypermetrope. In many cases it soon reaches a stasis and leaves only a slight irregular astigmatism to mark its demise. In others it is much more serious, leaving not only a slight cone, but great irregular astigmatism with myopia and accompanying low vision; while the exaggerated cases have a well marked cone protruding between the lids and almost blindness.

It is sometimes found in the rhachitic subject, but oftener in the nervous, chlorotic individual, and some fault with the general system is always present. The nervous phenomena common to puberty being the most important.

The specific ætiology is unknown. The intraocular tension is not above normal and the thinning of the cornea results from the increase in area and corresponding thinning of the membrane. There being a fault in the general nutrition of the eye, the tonicity of the cornea is insufficient to resist the

normal intraocular pressure, and hence the giving away of the least protected portion of the organ by the palpebra and extrinsic muscle of the eye, which is the point just below and to the inner side of the center of the cornea.

When the process has once begun the error of refraction resulting therefrom causing increased effort at accommodation, increases the progress of the disease, which, if the cause is not removed, hastens the eye to destruction.

When the disease is well advanced the diagnosis is easily made by the dark disc in the pupil reflex, the small and irregular corneal reflex or the irregularity of the rings in Placido's disc. When the ectasia becomes a staphyloma it can easily be seen, as it protrudes between the lids.

As the success in treatment depends on an early diagnosis which is very easily omitted, finer points in diagnosis must be found. As the center of the ectasiæ always occurs to one side of the optical center of the cornea, astigmatism is always present and also the meridians of least and greatest curvature are not at right angles. It is here that the ophthalmometer is invaluable. It not only shows the astigmatism, the variations from right angles of the principal meridians, but the variations with different stages of dilatation of the pupil and the course of the disease whether progressing, retrograding or at a stasis.

When vision and refraction are found to vary with different sizes of the pupil, accompanied by variation in the location of the axes of the cylinders, keratoconus should be suspected and can only be confounded with keratectasia, resulting from corneal lesions, the history of which is generally sufficient to determine the diagnosis, but when not, a little time and careful observation will do so.

With an early diagnosis and appropriate treatment, prognosis is good, the disease not only being brought to a stasis but considerable ectasia reduced to normal cornea. When more advanced, much improvement can be made, but vision will not be brought back to normal with correction of refraction, and ocular weakness will always remain.

When well advanced and the ectasia has become a perceptible cone, treatment yet is very valuable, but the preservation of good and comfortable vision is not possible.

Treatment should begin by correcting any errors in the general health and following good hygienic principles. Second, rest of the eyes from all use requiring prolonged accommodation. Third, correction of errors of refraction by appropriate lenses. Here care and judgment is required, as the refraction will vary very greatly with the different stages of dilatation of the pupil. The proper glasses should always be determined without the use of a mydriatic, care being taken to have about the same degree of light as in which the patient will work. Owing to contraction of the pupils in accommodation, sometimes very different lenses are required for distant and near use. In the early stages when the ectasia is progressing or retrograding, frequent changes of lenses will often be needed. Plus cylinders are usually but not always best.

In the early stages before the cornea has become very much thinned, local applications of the crystal of alum should be made to the affected portion of the cornea. After cocaining the cornea a smooth piece of alum should be gently passed over the cornea from one to half a dozen times, as experience teaches. This leaves a very soothing sensation and should be continued from once a day to two or three times a week till the ectasia is reduced or no longer improves. If after treatment is stopped the ectasia should return, the same treatment should be continued.

In the more advanced stage when the cone becomes visible and the center thinned, myotics and iridectomy are valuable but cauterization penetrating the entire thickness of the cornea is much more valuable, subsequently making iridectomy where it will give the best optical results.

To summarize: Keratoconus is often a self limiting disease. In the early stages the process can not only be stopped, but the resulting ectasia reduced and sometimes entirely removed, leaving a normal eye. When advanced the trouble can only be ameliorated and good vision never restored. When a high degree of ectasia has occurred, but poor vision at best can be preserved. Hence the importance of an early diagnosis and of acquainting the subject with the seriousness of the disease and of the importance of early and thorough treatment.

DISCUSSION.

DR. ALT.—What does the doctor expect from the application of alum, and in what proportion of cases has he seen a decided effect of it on the corneal tissue? I do not understand the possible effect. If it is used strong enough to harden the tissue of the cornea it would interfere with its nutrition and, perhaps, might produce sclerosis with beneficial effect. But I doubt even this possibility.

DR. SUKER.—I would like to ask the doctor if he considers keratoconus identical with keratoglobus, and whether it may not be a congenital condition. I would like to say that it is not always so, nor always binocular. Not much was said about the tension of the eyeball, or whether an iridectomy is of any avail. Would like to ask the doctor about his experience with eserine in these cases.

DR. O. A. GRIFFIN, Ann Arbor, Mich.—In reference to the correction of refractive errors in this condition, the doctor says that we should not use a mydriatic. It seems to me that in the weakened state of the cornea, the refractive condition should be as carefully estimated as in any other case, especially if the patient is young. The spherical aberration which is increased in these cases with dilatation of the pupil, may be counteracted by placing a disc before the eye with a perforation corresponding to the size of the normal pupil, through which the patient sees. An estimate of the refraction made in this manner brings out all the latent defect, with none of the disadvantages of mydriasis.

DR. WILDER.—When it comes to the point of cauterizing the tip of the keratoconus, it of course means that we will have a scar which will interfere with central vision. I had one experience where I had fairly good results by making a crescent-shaped cicatrix at the base of the keratoconus. The contraction of this cicatrix seemed to cause a flattening of the top of the cone, so that vision was markedly improved.

DR. BRADFIELD (closing discussion).—The discussion of this subject and questions asked confirm my supposition and give the opportunity to present to the Academy my views more fully.

My observation is limited to my own private practice, and

only having about 8,000 cases to draw from, I frankly admit that my conclusions should not be taken as final, but only ask that they be given their proper place.

In reply to Dr. Alts's question will say, that just how the alum affects the cornea I do not know; but I do know that in the proper cases under its use the ecstasia disappears as is demonstrated both by the ophthalmometer and the refraction. One case treated over a year ago in which vision was $\frac{20}{80}$ in the left eye, $\frac{20}{25}$ w.—150°—1.00 ax. 135°, the ophthalmometer showing the two principal meridians markedly at variance from right angles, now has a vision = $\frac{20}{20}$, accepts a plus 0.75 sphere, and has almost a perfect cornea shown both by the ophthalmometer and the refraction.

In answer to Dr. Suker's question, I will say I do not think keratoconus is ever congenital; I never saw a case before puberty, and do not think it related at all to kerotoglobus.

The conclusions from my observation on keratoconus which I wish to present to the Academy may be summed up as follows:

Keratoconus results from disturbance in the general system affecting the nutrition of the cornea, of which the nervous phenomena of puberty are the most important. Attention to the constitutional trouble is the most important part of the treatment. While the cornea is in this plastic condition applications of alum will reduce the ecstasia, but after the disease has come to a stasis it will be of no use.

When the ecstasia has advanced till it has become staphylomatous and the center thin, only a radical operation, as cauterization or excision, is of any use.

Many cases of keratoconus recover spontaneously before any perceptible cone results, but owing to the irregular astigmatism left, require special attention in correcting errors of refraction, owing to the different refraction of different portions of the cornea as the pupil varies in size. To be successful, treatment must be begun early and continued till the cornea recovers its proper nutrition and tonicity.

OPTIC NEURITIS (BILATERAL) COMPLICATING WHOOPIING COUGH.

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CHICAGO.

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A DILIGENT search into the literature has revealed reports of three cases of optic neuritis complicating whooping cough. I have thought it best to give the full report of these cases, in addition to report of my own case, and have also included an abstract of a case of ischemia of the retina, coincident with this disease, for reasons given below.

Alexander's¹ Case II: "Girl, 12 years old (still under treatment), totally blind, Oct. 3, 1887. Whooping cough preceded blindness two weeks and accompanied by intense headaches. On Sept. 15 the girl noticed that everything grew darker, and on Oct. 3 was totally blind. Pupils rigid, no reaction, either consensual or accommodative. Cornea, iris, nothing abnormal, but ophthalmoscope revealed in the fundus of both eyes optic neuritis.

Under treatment blindness decreased, and Nov. 1 was able to count the fingers at a distance of 8 inches. Middle of Nov., still better and decrease of optic neuritis. Whether there will be complete recovery, cannot be said on account of advanced stage of atrophy of the optic nerve."

Jacoby's² case I: "Girl, 6 years old, consulted with Dr. Hohlich, Nov. 15, 1888. Patient has had different diseases; when two years old, pneumonia accompanied by convulsions. From that time child complained of pain in the occiput. The other children in the family have passed recently through all the stages of whooping cough, and are recovering. For some weeks the child has suffered from spasmodic cough and occipital pains. The day before yesterday the child asked her mother why she remained away so long, as it was getting dark. This aroused the mother's attention that the child was becoming blind. Next day the patient was examined by Dr. Gruening. His findings were, dilatation of pupils ad

maximum; no reaction to light or convergence; bilateral neuritis without hemorrhages in the opticus; sensibility as to light quantitative. Another examination revealed that the vision was limited to right eye and that she was able to distinguish larger objects, like a watch, at a distance of 5 inches. Child examined again on 18th. Reaction of left pupil and larger objects could be seen with both eyes. Vision improves gradually. Was normal on Nov. 28. Ophthalmoscopic examination was negative. Since that time child is all right."

Callan's, P. A.,³ Case III: "Kate M., aged 11 years; patient undersized and not strong for her years. When 6 years old had a very severe attack of whooping cough, lasting three months. Patient was much reduced by severe whoops and mother feared for her recovery, she was at times so prostrated. At the end of three months of the disease the whoops suddenly ceased, but a very dangerous complication arose, viz., brain trouble. Patient on attempting to walk would become dizzy and stagger, complaining of severe headache and pains in the joints and all over the limbs. Mind wandered at times. Was obliged to remain in bed for three weeks and at the end of that time her headache and dizziness left her, but she could only see very imperfectly. The mother, who was not a very intelligent person, noticed that the child in walking would run against tables and chairs, showing plainly that she did not see well.

"Patient was examined by a very competent oculist, who told the mother that "the eye nerves were swollen" (optic nerves).

"For some months there was improvement in the girl's sight, but this failed her again. At the present time there is well marked white atrophy of both discs. V. R., movement of hands before the face, V. L., fingers at 8 feet.

"Here we have a case in which a long continued attack of whooping cough brought about a passive congestion of the brain, with edema. This led to choked discs and subsequently to atrophy."

Dr. H. Knapp⁴ reports a case of retinal ischemia in whooping cough in a boy 3 years old; total blindness, no hemorrhage in fundus or subconjunctival. Dr. Knapp be-

lieved the ischemia due to hemorrhagic effusion into sheathes of the optic nerves, or general anæmia. Paracentesis; improved vision.

Boy died three months later with pneumonia, as had been predicted by Prof. Loomis. This abstract of the case is reported because of the theory of pathogenesis advanced.

Author's Case:* Oct. 21, 1902, Ida B., aged 8 years, came in my service at the Illinois Charitable Eye and Ear Infirmary on account of subconjunctival ecchymosis of the right eye. Her mother gave me the following history: Four weeks before, the patient contracted whooping cough; has been whooping last two weeks. The coughing seizures, she says, are very severe; has six to eight during the night and fewer during the day. The mother says she is perfectly well excepting when she coughs. Has a good appetite; plays out of door as usual; sleeps well excepting when the seizures occur. She has never had any illness save an attack of measles two years ago, which left no sequelæ. She has never had convulsions. Does not complain of headaches excepting immediately after coughing spells for a short time.

The patient is the youngest of thirteen children, eight of whom are living and well; the others died of "lung fever" and other diseases at different ages. The mother is a well preserved, healthy woman. Father's health is good. The patient is a rosy-cheeked, well developed girl with no discoverable evidence of illness except during attacks of coughing. No motor disturbances to be found; possibly deep reflexes slightly exaggerated. Sense of hearing and smell normal.

Examination of the eye: R. V. $\frac{20}{15}$, L. V. $\frac{20}{15}$. Inspection of the right eye, aside from the subconjunctival ecchymosis, showed dilatation of the pupil, which responded to accommodation and consensually; to direct light very feebly. My colleague, Dr. J. Brown Loring, on making the ophthalmoscopic examination of the fundus, called my attention to the slight blurring of both discs.

Oct. 25. Patient presented herself, having ridden on the street car and walked together a distance of four miles. V. R. and L. $\frac{20}{15}$. Right pupil still dilated and responding feebly to direct light. Optic discs more blurred. Slight

*This case (by invitation) was reported at December meeting of the Chicago Pediatric Society.

amount of exudate in the retina below the disc, obscuring the temporal branch of the inferior branch of the central artery of the retina at one point. Analysis of the urine, both chemic and microscopic, negative. Temperature 99.2; pulse 80. Seems well.

Oct. 30. Ecchymosis gradually disappearing. V. R. and L. $^{20}/_{15}$. Right pupil responds to light better today. Neuritis more pronounced. Patient does not now have nor ever has had double vision. Temperature 99.2; pulse 80.

Nov. 6. No change in condition of the patient, excepting that neuritis is more pronounced, and temperature higher, 100° F.

Dec. 2. V. R. and L. $^{20}/_{15}$. Ecchymosis gone. Pupils respond normally to light and accommodation. Examination of urine, both microscopic and chemic, negative. Neuritis more pronounced. Mother says the child plays and acts in every way perfectly well. Whooping cough subsiding. Temperature same as at last visit, 100 F.

Jan. 15, 1903. Seems well, but has increased temperature, 99.5° F. Pulse 80; coughs occasionally; V. R. and L. $^{20}/_{15}$. Fields for red and green normal. Had difficulty in getting the peripheral fields on account of inability of the patient to appreciate the test; however both seem about normal. Discs still swollen; retinal pigment somewhat disturbed.

A blood count by Dr. E. V. L. Brown, Asst. Pathologist of the Illinois Charitable Eye and Ear Infirmary, and Dr. W. K. Spiece, was made with the following findings: "Reds," 4,966,800; "whites," 10,000. Therefore, whites to reds as 1 to 496.

The blood examination, as well as the general appearance of the patient, show that anemia is not the cause of the neuritis.

March 4. Temperature normal. Pulse 76. V. R. and L. $^{20}/_{15}$. Patient attending school; good appetite; apparently well—however has an occasional coughing seizure. Right disc slightly swollen, but evidently well on in regressive stage. The left disc slightly pale, but no swelling present.

May 20. Swelling of discs entirely gone; a perceptible amount of connective tissue at site of exudate on vessel described above; also a decided deposition of connective tissue in discs V. R. and L. = $^{20}/_{20}+$.

Prognosis in this case cannot be definitely known at the present time, but it is altogether probable that good vision will remain.

Analysis of findings in the above cases: A study of these four cases shows that optic neuritis occurs in girls, beginning about the 14th day of the convulsive stage in half the cases, opportunity for observing these cases being good; while in the other two cases the complication followed "after some weeks" and after "four months," the evidence being not so reliable.

Evidence of cerebral trouble—"intense headache" and "severe headache, dizzy, would stagger, mind wandered"—present only in half the cases. Ophthalmoscopic findings of the three authentically reported cases show optic neuritis without hemorrhage in the opticus. In only one case (author's) was exudate in the retina reported. In Dr. Callan's case ophthalmoscopic findings were not given except the expression "swollen eye nerves."

Disturbance in motility of the iris reported in all of the cases; in three vision was greatly reduced, while in the fourth there was no disturbance perceptible.

Perfect restoration of sight followed in one case (Jacoby's), while normal vision is present in author's case and but little disturbance of sight is probable. In Alexander's case, vision improving but no final report made; while in the Callan case white atrophy followed with quantitative vision.

Optic neuritis with and without cerebral complications, as above stated, suggests the probability of the cause not being the same in all cases. In sudden hemiplegia, aphasic disturbances, hemianopsia, etc., coming on during the coughing attacks, modern authors almost unanimously give the credit to "mechanical influences," that is, rexis, with the accompanying hemorrhage into the brain and cerebral meninges, and other circulatory disturbances.

The tetanic expiratory movement which characterizes the coughing attack in this disease, increases the intra-venous pressure to such an extent that rupture of the smaller veins and capillaries occasionally occurs, producing the above results, in the same way that sub-cutaneous and sub-mucous ecchymoses are seen in the skin and mucous membranes. The

optic "nerves" may become involved in such complications when meningitis ensues in the form of a descending neuritis.

In the above four cases reported, three of optic neuritis and one of ischemia of the retina, an attempt at giving the pathogenesis is made by Knapp and Callan only. Knapp explains the case of ischemia of the retina as being probably due to "hemorrhagic effusion into the sheaths of the optic nerves;" while Callan believes that "long continued attacks of whooping cough brought about a passive congestion of the brain with cedema; this led to choked disc and subsequently to atrophy."

In this connection it might be well to mention a case reported by Sebrigondi⁵ in which a girl of 6 years is said to have become blind with every coughing spell, produced, he thought, by blood stasis.

A. Steffen⁶ reports a girl of 8 years of age, who saw indistinctly during coughing spells and lost some of the sharpness of sight in the intervals while the spasmodic stage lasted.

Infectious influences: The consensus of opinion has not settled upon any particular germ as the cause of this disease.

Pronounced leucocytosis,⁷ more precisely speaking, lymphocytosis seems to be the only blood change so far observed.

Pathologic changes in the blood vessels have not been reported, I believe; however, the well known predilection that infectious diseases and toxic states have for the vessels, especially of the nervous system,⁸ render it possible that fatty changes occur in this disorder in the capillary endothelium of the vessels of the brain and the brain tract we call the optic "nerve."

This infectious disease is characterized by convulsive or spasmodic manifestations. Whether the infective agent excites the respiratory spasm through central or peripheral irritation or inflammation of the nerves supplying the pharynx, is as yet undetermined.

Peripheral neuritis does occur in this disease. Eschner⁹ has collected the reports of seven cases. Three of these, the cases of P. J. Moebius,¹⁰ E. Mackey¹¹ and M. L. Guinon,¹² unquestionably should be so classified. F. A. Craig¹³ reports a case which Eschner believes to be an inflammation of the sixth and seventh nerves.

The optic neuritis in the case I have reported is probably intra-ocular, for the especial reason that there has been no disturbance of central vision, no scotoma for red or green, and for the additional reason that peripheral vision is good. I think it is due to the direct action of the toxins of whooping cough upon the nerve tissue. The on-coming of the inflammation during the most acute period of the disease, the second week of the convulsive stage, and the gradual recession of both the neuritis and the spasmodic cough, might favor either the mechanical or the infection theory. If the cause is mechanical, the disturbance is exerted *through* the veins and not *outside* of them as in hemorrhagic extravasation or œdema of the optic nerve sheaths, for the reason that there has been absence of symptoms of brain lesion.

Blood stasis is but a temporary symptom, the equilibrium of the circulation soon being established. It would not account for this condition unless chronic local venous engorgement of the nerve head resulted therefrom, accompanied by diapedesis of the leucocytes, etc.

Such a condition can, I think, be better explained as resulting from the influence of the toxins according to Metschnikoff's theory of phagocytosis.

Generalizations derived from the study of four cases can be, at most, only suggestive.

Conclusions suggested:

1—Optic neuritis complicating whooping cough seems to occur most frequently in girls (four cases all girls).

2—Occurs with and without evidences of cerebral complications.

3—Vision may or may not be disturbed.

4—Prognosis as to sight good when no cerebral complications exist.

5—Optic neuritis may result from direct action of toxins of pertussis upon the nerve head.

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DISCUSSION.

DR. WILDER.—I saw this case when it came into my service at the Illinois Charitable Eye and Ear Infirmary in Chicago. The peculiar feature is that there should be this disturbance about the head of the optic nerve and yet so little functional disturbance of the eye. The central vision was normal and no contraction of the peripheral vision. The patient still has good central vision, so our only conclusion can be that there was not a very severe inflammation, although the appearance of the outline of the disc suggests beginning neuritis. In this case it was probably a slight œdema of the tissues of the disc and not an exudate, because vision was so good and there was no contraction of the visual field. The veins were somewhat engorged and tortuous. It was evidently a case of beginning neuritis; whether due to the toxins of whooping cough or due to the spasms of coughing is a surmise. We do know there is congestion of the vessels of the head in coughing, and we can understand how intraocular hæmorrhages may occur, particularly if the condition of the blood is such as to allow weakening of the walls. Slight œdema of the nerve or retina might be caused in the same way.

DR. GAMBLE (closing discussion).—In reply to Dr. Wilder I would say that the exudate entirely obscured the vessel above spoken of, at one point. The vessel is still obscured at this point to a lesser extent than it was three or four months ago. It has the appearance of having become organized into connective tissue. This is also true of the exudate in the heads of the nerves. Suggestions of bands of connective tissue can be seen. A few months later this condition will be easily demonstrated, I think.

NEURASTHENIC ASTHENOPIA.

BY L. J. GOUX, M.D.

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UPON investigation of text-books and literature on ophthalmology, I find the subject of neurasthenic asthenopia is given such sparse mention that one would be led to regard the disease as of very infrequent occurrence, or else one of such comparative insignificance as to be unworthy of much thought or attention. This has all the more perplexed me in view of the fact that my experience has brought me in contact with a considerable number of these cases. The importance of differentiating them from ordinary cases of ametropia or heterophoria at once becomes apparent, especially since we are at first apt to be misled by the patient whose history as told by herself would naturally lead one to the belief that some form of refractive error or muscular imbalance was the primary cause of the symptoms annoying the patient.

The diversity of results obtained, viz., only partial improvement or no improvement at all following the application of glasses or other treatment, led to a further investigation of this class of cases.

The term "neurasthenia" was invented by Beard in 1868. It is generic term applied to all morbid conditions essentially characterized by exhaustion of the nervous system.

According to the predominating phenomena, Regis has divided it into the following forms: cerebral form (cerebrasthenia), the spinal form (myelasthenia), the cardiac form (cerebro-cardiac neuropathy), the gastro-intestinal form (cerebro-gastric and intestinal neurasthenia), and lastly the genital type (sexual neurasthenia). Therefore it is seen that neurasthenia is not a disease but a group of diseases, a sort of diathesis with a most varied symptomatic expression.

The cause, which is essentially hereditary, takes its origin in the different diatheses, viz., neuroses, psychoses, alcoholism, syphilis, tabes, etc. It will be seen therefore, that in the large majority of cases degeneracy forms the ground work for the development of the malady. Also, the disease may be due to accidental causes such as shock, traumatism, etc.

As occasional disorders we have all the circumstances physiological or pathological, moral or physical, capable of either suddenly or slowly producing nervous exhaustion; puberty, troublesome pregnancies, disorders of the uterus or intestines, typhoid fever, hemorrhage, venereal diseases, sexual excess, mental strain, great fatigue, etc.

From this short description of the disease it can be seen how varied may be the manifestations of the disease. However, there are certain symptoms that are rarely absent, and they have been called by Charcot "neurasthenic stigmata." They are as follows: headache—frontal and occipital, sensation of emptiness of the head, insomnia and disturbed sleep, psychic adynamia, motor enfeeblement, spinal hyperæsthesia, gastro-intestinal atony, genital and vaso-motor disorders.

The symptoms referable to the head are almost invariably present and are usually of an aggravated form. Herein lies the reason for these patients being referred to or seeking the advice of the ophthalmologist.

Should the specialist not pursue his investigation of the case beyond the history of head symptoms as related by the patient, he will naturally be making the same error as was made by the one referring the case.

My experience with these cases has been somewhat as follows: Vision with or without mydriatic is $\frac{20}{20}$, or nearly so. Of course there may be some refractive error, but it is usually way out of proportion to the intensity of the suffering endured by the patient.

It is assumed that a mydriatic is employed as a routine practice in determining the full amount of error of refraction. One of the first expressions of the patient to put us on guard is the assertion that as the examination progresses there is a continued failure of vision, the patient complaining of fatigue and asking for periods of rest. In taking the field of vision we again note another peculiarity, viz., the longer we continue the test the more constricted becomes the field of vision. In endeavoring to accurately locate the axis of astigmatism, we here come upon another characteristic manifestation of the disease. There is a constant shifting of the axis of astigmatism, and this may be true whether actual astigmatism exists or not. The symptoms may point to the presence of

muscular asthenopia, and it is here that another characteristic manifestation of the disease is found. Employing the Maddox rod test, it is found that there is no permanent point of fixation of the streak, and often there is a characteristic swinging movement of the streak similar to the swinging of the pendulum of a clock. This movement may be confined to one side of the candle flame, but frequently it swings rhythmically from one side of the flame to the other. The two last named manifestations, viz., the variable astigmatism and the uncertain heterophoria, with the characteristic to-and-fro motion of the light streak together with the constricted field and early fatigue of the eye under examination, I consider pathognomic of neurasthenia, especially when there is found no refractive error or one so low as to be out of proportion to the intensity of the symptoms.

Color-blindness is said to be present occasionally, though I have never observed it in any of my cases. Patients may complain of dread of light with blepharospasm, lachrymation, neuralgia, etc., symptoms which point to a supersensitiveness of the retina. Oftentimes blurring of vision seems to be the most disturbing feature of the disease, and inasmuch as this renders impossible the continued close application to near work, patients complain most bitterly about it.

In a large percentage of these cases, further investigation will reveal the presence of some of the other stigmata as classified by Charcot. In other words the neurasthenic eye, as it may be called, is only a link in the chain of symptoms characterized as stigmata of this disease. These symptoms alone do not constitute neurasthenia, but as in other forms of degeneracy it is the sum total of all the different manifestations which present the typical picture.

Experience has shown that this disease is almost exclusively confined to young females, though I have noticed it a number of times in women undergoing the menopause. Its sister affliction, hysterical amblyopia, is often associated with it, in which case the diagnosis becomes much simplified.

Going beyond the assertion that these symptoms are due to degeneracy, I should regard them as being due to irregular, spasmodic stimulation of the centers controlling the functions involved.

There is no reason why neurasthenics should not be affected with refractive errors similarly to any other class of patients, and often they are greatly benefitted by a prescription for the proper glasses. However, considering the origin of true neurasthenia, which is essentially central, we are not justified in promising or expecting a cure.

Because of reasons given above, the only reliable test in determining the presence or absence of refractive error is found in retinoscopy, and then the full correction should be worn in case some error can be demonstrated.

As a protection to ourselves and in justice to the patient, she should be given a clear understanding of her condition and be dispossessed of the idea that glasses are to be a panacea for all her sufferings. It should be impressed upon her that rest from near work is the most essential element in establishing relief or a cure. Constitutional treatment may be highly beneficial, though vigorous physical exercise in the open air and gymnastics indoors in inclement weather, I think, will be found most efficient in caring for these unfortunates.

DISCUSSION.

DR. GRIFFIN.—This class of patients is interesting in its symptomology and variability of refraction. I have at present a case under observation which presents many of the symptoms that the doctor has cited. The patient is a professor in the University of Michigan, who through hard work has been rendered very neurasthenic. To point out a few of the peculiar things about him, he imagines that the ingestion of certain articles of food, e. g., rice, produces a torsion of one of the eyes; and at other times a peculiar sensation about the eyes indicates the presence of a fever. Although he has been tested several times for the torsion and rise of temperature, when these conditions have been supposed to exist, I have not been able to substantiate any such conditions. A test both with and without a mydriatic shows an emmetropic eye upon one side and but $\frac{1}{8}$ diopter of astigmatism in the other. The muscular condition varies from time to time, and a correction for his presbyopia does not prove satisfactory. He has consulted ophthalmologists of note in the East with

no results. The fact that he is becoming presbyopic may explain some of the difficulty; one day he feels the need of aid in near work, the next not. A great many persons are troubled in that manner when they begin to wear the presbyopic glass. Another peculiar symptom about my case is the fact that he says he sees two lenses before the eyes, and is always conscious of two fields, although the lenses were carefully centered. I hope some one may give me an explanation of this phenomenon. Of course it is not necessary to add that these cases are unsatisfactory to deal with. They must be handled with a great deal of care and patience.

DR. BRADFELD.—I appreciate the paper and wish to ask the doctor if in this class of cases, when the patients insist that they get no benefit from the glasses, he advises they should wear them constantly or at their pleasure?

DR. HECKEL.—This condition is exceedingly perplexing and calls for all the ingenuity a man possesses, inasmuch as the dynamic refraction varies from day to day and from week to week. It taxes the physician's patience and the patient's patience. It may occur at any age. I had a case recently in a gentleman 65 years of age; he consulted every oculist in the city, including myself, without relief. Sometimes, in spite of everything you may do, glasses are of no avail.

DR. GOUX (closing discussion).—Some neurologists say that these cases are practically incurable, and this I think is true in all cases having their etiology in degeneracy. Cases due to other causes are more amenable to treatment and may be cured or relieved by removal of the exciting cause. In regard to Dr. Bradfield's question, I find that even though I have instructed my patients to wear their correction all the time, if I pin them down to an actual statement I find they are not wearing their glasses as instructed. I take the precaution of telling them to put them on when they get up and to keep them on all day. Sometimes it will be quite a long time before a patient will be able to wear a glass and get any comfort out of it. I have had a number of cases where they could not wear the glasses, though they were correct. The variable condition of the patient, dependent upon the condition of the nervous system, makes the conditions so different from day to day that what you might prescribe as proper to-

day might tomorrow not be satisfactory. These patients will visit every oculist in town, and that is why I point out the necessity of making them thoroughly familiar with the character of their trouble. If they are made to understand this is not a local condition but a manifestation of a general disease, they will be much better satisfied.

DEGENERATE OCULAR CHANGES RESULTING FROM CONSANGUINITY.

BY LEE WALLACE DEAN, M.S., M.D.

IOWA CITY.

I N presenting to you a short paper on this very interesting subject my main object is to secure your opinions regarding the various points suggested. I would also like to hear of cases of degenerate conditions about the head in children, the result of consanguinity, encountered in your practice. A search in scientific literature has failed to aid me very much in this line of work.

The question of consanguinity in the first degree in its influence upon the central nervous system has often been discussed; its evil results are so apparent that they have resulted in the prohibition of such marriages in many countries. The general bad results have been so manifest that consanguineous marriages were prohibited even among some of the savage people.¹ The Choctaw Indians are divided into two great septs and no man dare marry in his own sept. The Indians do this because they believe it makes a stronger people. Numerous similar cases could be cited.²

Among civilized people consanguineous marriage has been prohibited not only by civil law but by ecclesiastical law.

The question as to whether consanguineous marriage does produce degenerate conditions or not, is very nicely stated by Dr. Talbot.³

I am sure there is no question today that if there is a perfect parent stock, and if the offspring are perfect, there can be no bad results from consanguineous marriage. How rare it is, however, to find today such a perfect condition. Liv-

ing as our ancestors have, in unnatural surroundings, and as we do today, has made a perfect stock a rare thing in the human race.

If on the other hand there is in a family some hereditary taint, the consanguineous marriage of the first degree simply doubles the tendency for the development of the hereditary conditions. Such a condition may have been latent for generations and the marriage of first cousins has so doubled the tendency that several of the children will show the same degenerate conditions. Several of the cases cited later in the paper will illustrate this. Atavism indicates that quiescent factors may be present for many generations that cannot be detected.

With the exception of the central nervous system we find the largest variety of degenerate stigmata in the eye. The reason for this is that the eye is really a specialized portion of the brain and is consequently subjected to the same influences as the central nervous system during its development.

The so-called degenerate stigmata found in other parts of the body are frequently considered to be the result of consanguineous marriage. There is no reason why those of the eye should not be so considered. It might be questioned, however, as to whether some conditions like retinitis pigmentosa ought to be placed in this category.

I became interested in this subject because, when several years ago I was investigating the degenerate conditions of the eye, I found a history of a consanguineous marriage of the first degree of the parents of many of the degenerate children.

Because a child has one or two degenerate conditions it is not considered a degenerate. These conditions are so common that the individual is only considered as a degenerate when several stigmata can be found. The number required differs in different schools.

Some of my cases have been so interesting to me that I take the liberty of mentioning them.

Case 1. Male, age 17. Parents, grandparents, uncles and aunts had no serious eye trouble or degenerate stigmata. Parents are bright, well-to-do people. Has three sisters, Bessie, age 15, who is partially blind; Effie, age 9, whose vision is good; and Grace, age 4, who is blind. Here are

four brothers and sisters, three of whom are partially or totally blind. He has three cousins, one boy and two girls, all in the same family. Of these, Louis, age 16, is almost blind; Oran, age 10, cannot count fingers; and the girl, age 13, seems to have unimpaired vision. The trouble in each case was retinitis pigmentosa. The fathers of the two families were brothers and the mothers sisters, and the fathers were cousins of the mothers. As a result of these two marriages we have in one family four children, three of whom are practically blind, and in the other three, two of whom are partially blind.

Case 2. Female, age 14. Father and mother have brown hair. Parents were related. There is no history of malformation or other degenerate stigmata in the family. The girl is very large but weak; she is not intelligent. Her hair is white; iris is a light blue. Examination of the eyes revealed nystagmus $V = \frac{6}{24}$. Fundi without pigment. Diagnosis, albinism.

Case 3. This is not a single case but the report of a family with six children. The father, Mr. V., and the mother were both dark complexioned. There was no history of any degenerate condition in the family. They were first cousins. They had but six children. Of these, two were dark complexioned and had good vision; the other four were albinos and had a vision $V = \frac{6}{12}$ or less.

Case 4. Female, age 16. Parents second cousins. Patient's teeth were imperfect; she had but three upper incisors, one root germ not having developed. An examination of the eyes revealed $V =$ fingers in 2 m. Her eyes were so small that she had to hold her lids open with her fingers in order to see. On the right side there was a congenital absence of the iris. In the left eye the cornea is elliptical, being 3 mm. wide in a horizontal direction and 2 mm. in a vertical direction. On this side there is a large coloboma of the iris, one-half of the lower portion being absent.

Case 5. Male, age 17. Parents are perfectly healthy. None of his ancestors have shown any signs of degeneracy so far as could be learned. His father and mother were first cousins. He has one brother and two sisters. One of his sisters is an idiot. She has microphthalmus, misplaced ears, and other signs of facial degeneracy. The patient is exceed-

ingly simple minded. His ears are large and placed at right angles to the head. The helix is deformed. His face is covered with a growth of silky hair. The lower jaw is exceedingly small and retrusive. He has no adenoids but always keeps his mouth open. Examination of the eyes reveals on the right side a microphthalmic eye with a coloboma of the iris and choroid. On the left side there was an apparent absence of the eye. There were small lids and a socket. No eye could be felt by introducing the finger in between the lids and feeling about in the orbit. The lower part of the socket was deformed. The union between the malar and superior maxillary bone had never taken place. There was a space one-fourth of an inch wide between the two bones. The patient was given an anæsthetic and a dissection of the contents of the orbit was made. The fissure between the malar and the superior maxillary bone was found to extend back to the spheno-maxillary fissure. In the apex of the orbit lying closely against the optic foramen was found a rudimentary eye. It was removed. It was about one-third of an inch in diameter. With the naked eye no change indicating the cornea could be detected. The eye was hardened in formaldehyde and alcohol and stained with hæmatoxylin and eosine. The eye was composed of an outer thick fibrous coat. The cornea could only be differentiated microscopically from the sclera by the absence of an inner lining of pigmented cells. The eye was filled with myxomatous tissue much more solid than the normal vitreous. It contained some blood vessels, one of which was very large. No trace of retinal elements could be found. The internal structures seemed to have all undergone a myxomatous and mucoid degeneration. The lens and retina seemed to be absent.

Case 6. Female, age 5. No history of any serious eye trouble or of degenerate conditions in family. Parents were cousins. Child was very dull. Left eye began to enlarge shortly after birth. O. D. apparently normal, O. S. very much enlarged. Tension normal. Diagnosis: congenital glaucoma, Left. The left eye was enucleated. The usual cupping of the disc was present. Microscopical examination failed to reveal any obstruction of the canal of Schlemm.

Case 7. Female, age 6 months. I saw in consultation

with Dr. Cooling of Wilton Junction, Iowa. Parents were healthy; no history of any tumor or eye trouble in the family could be elicited. Parents were first cousins. The parents said that two months before the right eye began to increase in size, and a few weeks later the left began. The child apparently could not see. Pupils were dilated; would not react to light. Tension in right eye +2, and in left +1. Both eyes were enlarged, the right the more. Lying behind the iris in each eye could be seen a large tumor. Diagnosis: double sided neuro-endothelioma. Four months after the child was seen it died.

Case 8. Male, age 4. Parents' and grandparents' history negative. Parents cousins. Boy has congenital coloboma of iris in each eye and anterior polar cataract in the right eye.

Of 181 children in the Iowa College for the Blind in 1900, nine, or about 5 per cent., were the result of consanguineous marriage of the first degree. The number of consanguineous marriages of the first degree in the state of Iowa is far below $\frac{1}{2}$ per cent. I ought to say that it is exceedingly difficult to secure a history of consanguineous marriage when present. There were probably others present the result of consanguineous union.

If we exclude from the list those blind children who were blind because of blennorrhœa neonatorum, sympathetic ophthalmia, trachoma, etc., and consider only those who suffered because of congenital conditions, we would find that 14 per cent. were the result of consanguineous marriage of the first degree. These figures seem large. I do not think they ought to be considered as indicating the relative proportion of degenerate eyes in families the result of consanguineous marriage and in those not, because we are considering here only one field. Among the pupils who have entered the college since 1900 the per cent. is about the same.

The tendency for the increase of eye trouble amongst civilized races because of excessive use of the eye has had a tendency to increase the bad results of consanguinity on the eye. The Indians have very little eye trouble. About 40 per cent. of the civilized people have eye trouble due to disease from use. This may be a beginning change in the eye for the better. Fuchs believes that the individuals with the school

myopia have their eyes adapted to their work and that the eye is superior to an emmetropic eye. The eye is not perfect, it is changing. The socket is becoming less deep and the superciliary ridges less marked.

The influence of consanguinity on retinitis pigmentosa has been mentioned by several. Dr. Liebreich says that of 26 cases of retinitis pigmentosa, 53.8 per cent. were the result of consanguineous marriage. Magnus reports 33 per cent. of cases of retinitis pigmentosa in children the result of consanguinity. Of 66 cases reported by Chipault, 45 per cent. were the result of consanguineous marriage. Of 18 cases under my observation where the history of the parents could be secured, eight, or 44 per cent., were the result of consanguinity.

Retinitis is a disease that has a great inheritability. I have under observation a grandfather, mother and son, each having the disease. As it is a comparatively rare condition, there is not the chance for doubling the tendency of its appearing by marriage of individuals not related as the more common conditions.

A very careful examination into the history of the ancestors of the cases I mentioned failed to reveal any eye trouble of any importance. Yet in the first group mentioned we have these cases appearing by intermarriage in the two groups when consanguinity took place.

Albinoism is considered by zoologists to be a degenerate condition. Davis⁷ says that consanguineous marriage leads to albinoism. Certainly a large per cent. of albinos are the products of consanguineous marriages.

As to whether consanguinity may play an important part in neuro-endothelioma and congenital glaucoma can only be determined by hearing from more cases. The cases of microphthalmus coloboma of iris, congenital cataract, and anophthalmus mentioned are examples of arrest of development. It is interesting to note that the conditions of the eyes are just the opposite to those of the eyeless fishes and worms that have lost the use of eyes that were in their ancestors perfectly developed, owing to generation after generation living in the dark. Wagenmann⁸ has found that in these the lens and retina are almost the least of the structures acted upon. That the phyletic degeneration does not follow the reverse

order of development. None of the adult degenerate eyes resemble stages of past adult conditions. In the degenerate eyes, however, we frequently find the eye in one of its developmental stages.

The condition of the eye is not due to local conditions but is due to some central disturbance. With our present knowledge of the physiology of the central nervous system, one cannot say as to whether there are developmental centers—that is, centers which control the growth of parts—or whether there are simply the trophic centers which control their nourishment. The condition is due to a disturbance of the trophic center, or of both if they exist. One may expect these degenerate conditions in the products of consanguineous marriage, because of the increase of some hereditary tendencies in the germ.

By⁹ means of the nuclear division and formation of the second polar body, the excessive accumulation of different kinds of hereditary tendencies or germ plasms is prevented. With the removal of the second polar body, as many different kinds of idioplasm are removed from the egg as will afterwards be introduced by the sperm nucleus. If the sperm nucleus contains the same hereditary tendencies as the ovum there will be a greater tendency for these tendencies to become manifest than if the latter were different.

¹ Algonquins, Iroquois, Delaware, Canadian Indians. Huth's *Marriage of Near Kin*, p. 92.

² Huth, p. 93.

³ Talbot, *Degeneracy; Its Causes, Signs, and Results*, p. 79.

⁴ Liebreich, *Deutsche Klinik*, Feb. 9, 1861.

⁵ Chipault, *Etudes sur les. Mar.*, pp. 58, 59.

⁶ Magnus, *Die Blindheit*.

⁷ *American Medical Bi-weekly*, Vol 12, No. 13.

⁸ *Archiv für Entwicklung Mechanik der Organe der Menschen*, Vol. 8, No. 4.

⁹ Ribot, *Heredity*, p. 366.

DISCUSSION.

DR. VAIL.—This is a very interesting subject and one that is very important. I rise to report that out of seven children in two families where the parents are cousins, I have found five who had congenital cataract. Of the remaining two, one had slight hypermetropia and the other had

hypermetropia of six or seven diopters. In one family there were but two children. The first, a baby, had cataract and the eyes were operated on in the usual way. I enquired of the mother whether she and her husband were blood relatives. She denied that they were. A year and a half later another baby with congenital cataract was brought by the same mother. I asked again if she and her husband were not related, and she confessed it was true, they were first cousins. She said her husband told her the other time she must deny it, for fear of trouble in the courts. I felt it a duty at that time to tell her that I considered it a social crime for her to bear any more children by this man, and so far as I know they have had no other children.

DR. ALT.—I had occasion to observe congenital cataract in two children out of four in a family where the parents were first cousins. At the same time I also operated on the grandmother for senile cataract. This was, I think, an additional proof that it was due to consanguineous marriage. I have operated on a number of occasions in a large family in southern Illinois who have, for I do not knowhow long, intermarried. There is a large number of cases of senile cataract and also congenital cataract in the family. The members of this family are so much accustomed to this condition that one of them, who is a physician, comes to me every few months to have me look at his lenses and see if they are beginning to show cataract formation. He told me that his uncle whom I operated on last was, if I am correct, the 43rd case in the family that had been operated upon for cataract.

SARCOMA OF THE CHOROID.

By W. STANLEY SAMPSON, M. D.

LANCASTER, OHIO.

THE clinical history of intra-ocular tumors published by von Graefe in 1868 was so complete and exhaustive that little remained to be accomplished by his followers. It is not the aim of the author to break the seal placed upon the subject by the illustrious savant, but to content himself with an epitome of his classical monograph. Sarcoma of the choroid

is considered a rare disease, and occurs most frequently between the fortieth and sixtieth years, seldom occurring in childhood—differing in this respect from glioma.

Four stages of the disease are distinguished. From the small tumor of the first stage, recognized only by ophthalmoscopic examination by detachment of the retina, to the second stage—that of increased tension—the transition may be sudden. The symptoms of this stage correspond so completely to those of inflammatory glaucoma, that a correct diagnosis is made with the greatest difficulty, and in many cases not at all. Pain is a cardinal symptom in this stage of the disease, and usually sounds the first alarm of trouble to the patient.

The third stage consists in the tumor passing through the sclera and its growth upon the outside. At this period the pain ceases, and the orbital cavity is filled more or less rapidly, depending upon the point of exit. If the sclera is ruptured posteriorly, the ball is pushed forward, and the growth is longer in making its external appearance than were the rupture anterior or lateral. The growth is now more rapid and the tumor may become as large as the fist.

The fourth or metastatic stage of malignant tumor of the choroid is primary in nearly every case, but generalization of the tumor by the development of metastatic nodules in other parts of the body may occur, and especially in the liver.

“Manz reports a case involving both eyes, the original growth developing in the breast.”

The prognosis is grave and always proves fatal if the eye is not removed early. Berry gives a patient with sarcoma of the choroid, unmolested, five years to live. Of the 285 cases reported on by Fuchs, 13 per cent. recurred, and most of them in a year.

The treatment is enucleation as soon as the diagnosis is certain that the growth is confined to the ball, and exenteration if the orbital cavity is invaded and the whole growth can be removed.

Mrs. M. A. H., 67 years old, farmer's wife, family history good, consulted me on Sept. 20, 1901. Seven years ago she suffered severe pain in the right eye, and was treated by her family physician for neuralgia, but without benefit. One year

later she became blind in that eye. The pain suddenly ceased, and some time during the winter of 1900 she noticed a growth appearing at the inner canthus of the right eye. On examination I found a dark red mass, filling the orbital cavity and projecting far beyond the frontal eminence. The eyeball formed the outer portion of the growth, and by careful inspection indistinct traces could be seen of the iris and pupil. The growth was immobile and quite firm to the touch. On Oct. 2, 1901, I exenterated the contents of the

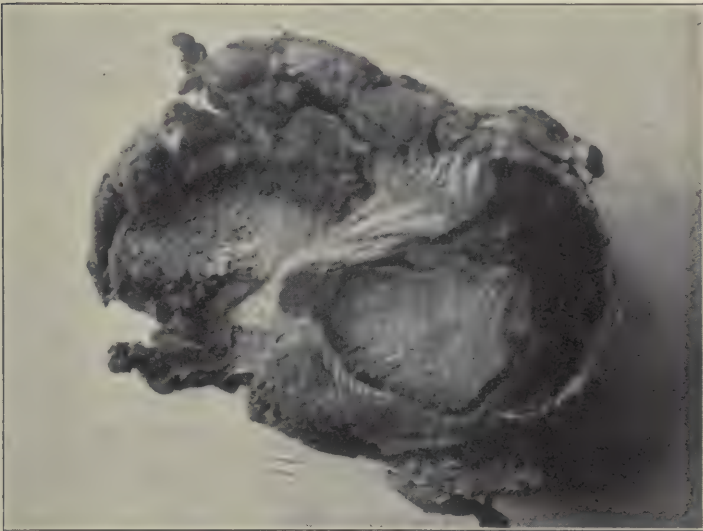


FIG. 1.—Sarcoma of the choroid. Growth actual size. The right hand portion of the tumor contains the eyeball; lachrymal gland resting on top.

orbital cavity. The growth after removal measured 5 x 5 x 7 cm., was highly pigmented and vascular. (See figure). The optic nerve and the muscles were consumed in the sarcomatous process, excepting the external rectus, which was held by a few remaining fibres to the optic foramen. The remaining portion of the optic nerve was caught by forceps and severed by curved scissors as far back as possible. After a thorough curetment the cavity was dusted with nosophen and packed with sterile gauze. The repair process was rapid and at the end of ten days the patient was discharged. There was no recurrence of the disease, but the patient died fourteen months later from metastatic involvement.

According to the patient's statement, pain preceded blindness in the eye one year, differing in this respect from the typical clinical history found in the text books on ophthalmology. A section through the growth bi-sectioning the eyeball, gives a clear idea of the point of rupture. A microscopical examination of a section made from the lachrymal gland shows round and spindle cells, pigmented. A section from the optic nerve shows a predominance of spindle cells.

Only recently my attention was called to the following interesting case: C. T., aged 26; excellent family history. When 15 years of age he was struck in the right eye with an arrow, made from an umbrella wire, thrown from a crossbow in the hands a playmate. There was no perceptible wound inflicted, and the eye caused no trouble until two years after, when he noticed that vision began to fail in that eye. Five years ago dark nodules began to appear on the superior surface of the ball, which seem to have pushed their way directly through the sclera. The growth is firmly attached to, or in other words is a part of, a tumor mass filling the eyeball, and when the nodules are viewed separately resemble coffee grains. The relationship of the iris is not interfered with, but the growth can be seen in close proximity to the iris by means of a strong lens and oblique illumination. The patient has been totally blind in this eye for two years. At no time has he experienced pain in or about the eye, and there is not the slightest tenderness on pressure at this time. This case has every symptom of sarcoma, save pain, which, as above stated, never was present. Glioma can be excluded by the patient's age and the pigmentation. We do not purpose going into the etiology of this disease, but consider the pathologic lesion in this case produced by trauma. Operation was refused and our diagnosis cannot be verified by microscopic examination, but the clinical history is at such a variance with that of the usual case that we venture this report.

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DISCUSSION.

DR. VAIL.—Within the past six months I have removed the entire contents of the orbit, including the eyebrow, eyelids, lacrimal gland, sac—in fact all the orbital structures, and while it seemed a most heroic operation, it was one of the easiest I ever attempted. I was surprised to find out how easy it was to shell out the orbital contents. The incision was simply carried around the outside to include the whole neoplasm, down to the bone, through the periosteum; then by means of the raspatory the periosteum was stripped up and followed around. It stripped off so easily it almost fell out. You just sweep around with your periosteotome, like taking a pie out of a pan, then with the scissors clip off the impacted mass of tissues in the apex of the orbit.

DR. ALT.—If I understood rightly, the author said that in the secondary stage there is always detachment of the retina. Twenty-five years ago this was held to be correct, but it has been proven untrue. In some cases there is detachment of the retina, in others the retina and growth are grown together, and consequently no detachment can occur.

DR. BRADFELD.—I wish to suggest that in cleaning out the orbit it is always safest to have the thermal cautery handy when the apex is reached. I would ask the doctor how he covers the orbit after the operation.

DR. VAIL.—In regard to the dressing, I simply packed the orbital cavity. The hæmorrhage was insignificant. It commenced, but we put in a packing of bichloride gauze, and the hæmorrhage stopped. I would hesitate to use the galvanic cautery or the chromic acid in the apex of the orbit, as I regard either to be dangerous—it is so close to the brain. It was the purpose of the after-treatment to be especially careful not to introduce germs from the outside. The dressing was of bichloride gauze, left for several days. Granulations gradually formed and it took four or five weeks for the periosteum to form and epitheliate. It was not necessary to do plastic surgery.

DR. WILDER.—It is easy to strip off the periosteum, as Dr. Vail says. I feel better satisfied, as Dr. Bradfield says he does, when I have the cautery with me. In one case I had severe hæmorrhage. By gently touching the bleeding point

with the galvanic cautery at a dull red heat you can sear these vessels perfectly, and I do not think such treatment is dangerous. Sometimes these cases, where exenteration of the orbit has been performed, will take a long time to heal. I have taken long ribbon strips of Thiersch grafts and with them lined the orbit, thus concluding the case more promptly. By waiting for the new epithelium to form, the healing process is much longer, and it seems to me there is greater danger of recurrence in the low grade granulation tissue.

DR. SAMPSON (closing discussion).—It is hard to describe this operation, but it is very easy to do, as Dr. Vail says. It was necessary to make a canthotomy on account of the size of the growth. In this case we controlled the hæmorrhage with sterile gauze. Skin grafting was not necessary in this case. I was prepared for hæmorrhage, but there was no more than we could expect in enucleation. If the lids are in a healthy condition, it is advisable to save as much as possible.

SOME REMARKS ON THE INFLUENCE OF ENVIRONMENT ON THE EYE.

By HAMILTON STILLSON, M. D.

SEATTLE, WASH.

MR. DARWIN, in his "Variations of Plants and Animals Under Domestication," quotes Dr. Lucas' remark that "there is not one single faculty of the eye which is not subject to anomalies." This statement Mr. Darwin proceeds to verify by quoting a list of cases of various anomalies transmitted, organic and functional—such as, for instance, in the first class, cleft iris, albinism, muscle anomalies, etc., and in the second class, color-blindness, night-blindness, day-blindness, etc.

What we need to remember is that inherited and transmitted anomalies are but accumulated acquired traits. These traits, usually so slight as not to be noticeable, become emphasized and magnified by transmission. Natural selection and adaptability to environment often work together.

This is beautifully seen in the rudimentation of the eye. The rudimentation of the eyes of the mole, for instance,

is due partly to non-use of the eyes, and partly to the inflammation of the eyelids or nictitating membranes from the presence of dirt. And, "as eyes are certainly not necessary to animals of subterranean habits, a reduction in their size, with adhesion of the eyelids and a growth of fur over them might in such cases be an advantage, and natural selection would aid the effects of disuse." So, too, with the eyes of the cave dwellers. "In some of the crabs the foot-stalk remains though the eye is gone"—clearly a rudimentation from disuse.

In the case of the cave rat, the *neotoma*, specimens have been captured a half mile from the daylight that have had eyes lustrous and large, but blind. It has always seemed to me that the opponents to the belief in *asthenopia ex anopsia* had not made the acquaintance of the *neotoma*. The evidences of these graduations of the effects of environment on rudimentation is remarked by Schiodte. "We accordingly look upon the subterranean fauna as small ramifications which have penetrated into the earth from the geographically limited faunas of the adjacent tracts, which, as they extend themselves into darkness, have been accommodated to surrounding circumstances. Animals not far remote from ordinary forms prepare the transition from light to darkness; next follow those that are constructed for twilight, and last of all those destined for total darkness, and whose form is quite peculiar."

Thus, from Schiodte's point of view, by the time an animal had reached, after numberless generations, the deepest recesses, disuse will have more or less perfectly obliterated its eyes. This view is held by Lyell, by Agassiz, who was the first to describe the *amblyopsis*, and in fact by all naturalists so far as I know.

If these acquired traits may be transmitted, would it not be well to impress upon our minds the importance of the effect of daily environment upon the eye, such effects as come from occupation, climate, habits, etc.? The eye in its embryological development is formed by a juxtaposition of two saucer-shaped layers. The eye, then, is, as a rule, at birth hyperopic (perhaps astigmatic), and over-use of the eye in early childhood tends to deform it more. The statistics looking towards the effects of occupation upon the pro-

duction of myopia are not yet sufficiently studied to warrant a definite conclusion, but we might say in general terms that the prolonged use of hyperopic or emmetropic eyes at near vision will tend to the production of myopia. The infrequency of myopia among watchmakers may be accounted for by the fact that as a rule watchmakers use a microscope before one eye in viewing the object manufactured.

But the prevalence of myopia among students is proverbial. The effects of school life upon the eyes are dwelt upon at length in the text books, and we may safely relegate this part of the subject to the text books. But I cannot refrain from quoting one or two pregnant remarks. Risley, in Norris and Oliver's "System of Disease of the Eye," sums up the production of myopia in school life by saying: "The obvious association of the increasing percentage of myopia with the work of the schools seemed naturally to fix the responsibility for the disease upon the educational process, and led directly to efforts for the discovery and reform of faulty educational methods." And again, "The obvious lesson is that our children enter upon their educational training at a too tender age, and that during the first years at school the methods of instruction should be so modified as to avoid as far as possible continuous work at a near point."

The manner in which miners acquire nystagmus has been well observed. The semi-darkness of the mine, the semi-recumbent position of the miner with his head diagonally upward, and the constrained position of all the muscles including those of the eye, produce such a want of harmony in the ocular muscles as can only be expressed by nystagmus.

I, myself, have observed that among the old school potters there usually exists a high degree of hyperphoria. The position of the potter at his lathe is semi-recumbent; he stands upon his right foot with his body leaning upon a back-rest. His left foot is pressed against the tread of the lathe, while his head is bent forward toward the right and his eyes look into the vessel that is being turned, and into which his right hand is inserted. This produces habitual torticollis and a hypertrophy of the lower left rectus and the right upper rectus. A right hyperphoria of 4 degrees is not uncommon among such potters.

I seem to have noticed also a great prevalence of high degrees of astigmatism among the Sisters of Charity. These nuns usually wear a head-dress shaped like an old-fashioned sunbonnet, the front part projecting from four to six inches in front of their faces, and is lined with white. With such blinkers on the Sisters can only look straight forward or demurely down. There is, therefore, little lateral pressure against the eyeballs, and if the Sister has entered the service early in life, high astigmatism seems inevitable. At any rate, in my observation astigmatism is quite prevalent among them.

We are all familiar with the type of ocular fatigue produced by a change of residence from the country to the city. The visitor from the country unaccustomed to glancing rapidly from side to side at closer range than usual soon fatigues the ocular muscles, producing a nausea somewhat similar to that produced in some cases by a ride upon a street car.

A similar condition is often brought about by the amateur typewriter. The frequent movements of the eyes from the copy to the keys of the finger-board soon produce incoordination. This confusion seems to be produced more rapidly if astigmatism exists, and if the keys be round. Some manufacturers of typewriting machines have noted this fact and now manufacture their machines with octagonal or square keys instead of round ones.

Many other disturbances of the eye from occupation will doubtless occur to you.

I wish to refer to the effect of change of climate upon the eye. I seem to have noticed that persons who have migrated from the south to the north have much more difficulty with their eyes than persons who have migrated along the same parallel of latitude. The population of the Puget Sound district, for instance, is very cosmopolitan; many of the inhabitants are from the southern states, many of them from Mexico. Many of them, indeed, migrate in the summer months to Alaska and return. The effect of this north and south migration, especially during the first few seasons, seems to be productive of retinal congestion, or optic neuritis. This, of course, would be particularly the case if the patient suffered from snow-blindness during his visit to Alaska, and

the neuritis would then be accompanied by conjunctivitis and corneitis.

Another peculiarity is the effect upon these patients of a change from a sunny clime to a cloudy one. The north Pacific coast is foggy and humid. This humidity seems to chill the surface of the body, preventing perspiration. To a person accustomed from infancy to rapid perspiration (such as would be produced by a warm, sunny climate), there would exist a demand for increased activity from the kidneys, and the demand is often greater than the kidneys can comply with. In consequence of this the kidneys often become inflamed and unable to perform their function. Retained products of waste very frequently produce retino-choroiditis and optic neuritis. So, too, in regard to the use of the eyes in this foggy atmosphere; a person accustomed to bright skies finds it difficult to use the eyes at prolonged near work in a climate whose skies are usually overcast by cloud. So that while persons from Scotland, Ireland or Scandinavia have very little difficulty in accustoming their eyes to the climate of Puget Sound, those from the Mediterranean, the southern states and Mexico, have great difficulty in doing so. I ought to observe, however, that the Scandinavians in migrating to the northwest country suffer degeneration in other ways. Dr. Ivar Janson, in the January number of the *Northwest Medicine*, gives a very graphic account of his observations upon his countrymen. He mentions the fact that while confined to their mountains in northern Europe these people live an Arcadian life, becoming vigorous, large and well proportioned, remarkably free from disease. Yet upon migrating to the northwest there is a rapid degeneration physically and mentally. "The proportion of Scandinavians in the hospitals for the insane is quite appalling." "And tuberculosis * * * seems to make up for lost time for past immunity. * * * I give tuberculosis twenty-five per cent. of all the dead of the first descendants of people whose environments and means of sustenance for untold generations had formerly been stable and fixed."

I wish to observe also that persons migrating from the southern Pacific coast to the northern Pacific coast experience at first a peculiar somnolence. In my own

case, while a resident of California, five hours' sleep each night seemed ample, and indeed during the summer months three or four hours' sleep nightly was all that was obtainable on account of the heat. But at Seattle nine hours' sleep nightly seems indispensable—ten hours desirable. This "letting down of the nerves" has a peculiar effect on the habits. At Victoria the older business houses open at 10 A. M. and close at 2 P. M., and the major portion of the time is spent in eating and sleeping. A noteworthy change is observable in women. Women who in California are unable to conceive usually become quickly pregnant upon migrating to the Puget Sound country. Women seem, however, to reach the menopause early in the north, often as early as the thirty-fifth year, though girls are slower in reaching puberty. Puberty is sometimes delayed until the twentieth year. These changes in the "habits of the nerves" have a profound influence on the eyes, inducing among other phenomena very early manifestations of presbyopia.

In the far north—in Alaska for instance—the monotony of the long dreary nights in winter induces profound neurasthenia, particularly in women; and in all classes prolonged confinement indoors, and continued efforts at reading to kill time, make their injurious impression on the eyes.

Not to extend this paper unduly, let me close with the remark that what I have said has been said not to exhaust the subject but to call forth a discussion.

RARE OCULAR LESIONS IN SCARLATINA.

By ELLET O. SISSON, M.D.

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Member Ninth International Ophthalmological Congress.

THE ocular lesions that occur in scarlatina are of particular interest for the reason that they are generally of a more or less serious nature. Scarlet fever, unlike diphtheria, is a disease that the average specialist does not come in contact with. It is handled in nearly every case by the general practitioner, and it is only when the eye symptoms are of a prominent character that a specialist is called in, and as a result the connection between many eye diseases and scarlet

fever is lost sight of. Among the rarer lesions we have uræmic amaurosis, purulent inflammation of the vitreous, phlegmon of the orbit, and thrombosis of the cavernous sinus.

In uræmic amaurosis, which occurs more frequently than the other lesions, the blindness comes on suddenly and gets to be complete within a few hours or a day. After one or more days the sight is gradually restored.

Simultaneously with the attack of visual disturbance other nervous symptoms exist, such as headache, vomiting, dyspnœa, loss of consciousness and convulsions. The fact that the reaction of the pupil to light is in most cases preserved in spite of the complete blindness, proves that the location of the affection cannot be in the eye or in the optic nerve, but higher up—that is, in the brain, which is poisoned by the excretory matters contained in the blood. Cases are reported by Ebert,¹ Förster,² Monod,³ Power,⁴ Loeb⁵ and Martin⁶. These cases were quoted by Förster,⁷ who called attention to the fact that in all of them albuminuria was present, and that the amaurosis occurred in the desquamation stage after a period of generally favorable symptoms. The amaurosis was ushered in by cerebral symptoms, headache, convulsions, vomiting and stupor. It came on suddenly, was bilateral, and for a time was complete. No ophthalmoscopic lesions were detected, and the blindness gradually cleared off.

Purulent inflammation of the vitreous or suppurative hyalitis is the product of a metastatic choroiditis, which sometimes follows in the wake of scarlet fever. In this lesion if the cornea is clear, a yellowish reflex is seen shining through the pupillary space, there is retraction of the periphery of the iris and bulging of its pupillary border. Usually one or two synechiæ are present and the tension is diminished. In addition to this, there may be a pericorneal zone of congestion connected with the inflammation of the iris and ciliary body.

When the pus in the retina is circumscribed the symptoms at the first glance are not unlike those of glioma of the retina, and the name pseudo-glioma has been given to this condition, especially as it is seen in children.

In phlegmon of the orbit we have an inflammation of the cellulo-fatty tissue surrounding the eyeball. It is of metas-

tatic origin and is generally of the acute form. It is usually monolateral, although it may be bilateral. Constitutional symptoms generally accompany it, such as chills and fever, and not infrequently cerebral symptoms, such as headache, vomiting, mental hebetude, retardation of the pulse, etc. The local symptoms are exophthalmos, limitations in the movements of the eye and swelling and œdema of the lids, together with hyperæmia and chemosis of the conjunctiva. When the symptoms have reached their acme the skin of the lids at a certain spot grows red, then shows a yellow discoloration, and finally is perforated by a discharge of pus. After the evacuation of the pus, which is present in large quantity, the inflammatory symptoms in most cases rapidly subside and the opening soon heals again. The sight may suffer permanent diminution or be altogether annihilated, if the optic nerve is implicated, for, inflammation of the optic nerve or thrombosis of its vessels may develop, succeeded by atrophy of the nerve. Detachment of the retina and even panophthalmitis, also, occasionally occurs in retrobulbar phlegmon. If the suppuration is carried over from the orbit to the cranial cavity it leads to a fatal issue through purulent meningitis or abscess of the brain.

In thrombosis of the cavernous sinus we have a lesion which from the pathologist's view-point is of particular interest. That the lesion is a rare one is shown by the literature on the subject, there having been only 182 cases the result of various causes reported up to date. Of this number only fourteen recovered. The lesion is set up metastatically. The symptoms are very prominent and are similar to those which present themselves in the beginning of a retrobulbar phlegmon. The lids and the conjunctiva swell up with œdema, and the eyeball is protruded and becomes hard to move. The veins of the retina are seen, upon ophthalmoscopic examination, to be distended enormously with blood. These symptoms are referable to the fact that the veins of the orbit discharge the greater part of their blood through the ophthalmic veins into the cavernous sinus; if the latter is occluded an extreme degree of venous stasis in the orbit necessarily takes place and leads to protusion of the eyeball and also to venous hyperæmia of the retina. At the same time there

may be a doughy œdema in the mastoid region. This œdema depends upon the fact that in this region an emissary vein of Santorini empties into the transverse sinus and thus indirectly into the cavernous sinus, so that when there is occlusion of the latter this region also shares in the venous stasis. When this œdema is present it forms an important diagnostic sign between thrombosis of the cavernous sinus and retrobulbar phlegmon, in which latter it is absent. A further point of difference lies in the fact that thrombosis of the sinus frequently passes over to the other side, so that the same complex of symptoms develops there also, while, on the contrary, a bilateral orbital phlegmon would be one of the greatest rarities.

Wells and Germain⁸ report a case that was operated upon, the cavernous sinus exposed, incised and drained. The patient died, but they claim that by the operation they demonstrated that the sinus is not inaccessible, that it may be reached without grave danger to the patient, and at least a low mortality from the operation itself, that it can be done under almost primary anæsthesia, not associated with any degree of shock, finished within a few minutes—in their case eight—and that the hæmorrhage is easily controlled. They also claim that an incision into one sinus instantly and completely relieved the interference with the circulation in both.

Retinitis albuminurica occurs after scarlatina, but is not frequent; it is more a complication of the chronic form of Bright's disease than of the croupous nephritis which is found in scarlatina. The prognosis is more favorable than in albuminuric retinitis unconnected with an exanthem, but partial optic atrophy has been observed⁹.

The sight can also be affected as the result of scarlatina without the presence of albuminuria or any evidence of any kidney disease, as in the cases recorded by Hodges¹⁰, where thrombosis of the central artery was observed in one eye of a growing girl while recovering from scarlatina. In this case the urine was normal, so that the ocular lesion may possibly be attributed to debility induced by the disease.

Pflüger¹¹ has observed papillo-retinitis after scarlatina without kidney affection, and the same phenomenon has been observed by Betke¹² though in the latter case the patient had

suffered at an earlier period from hemiplegia. Leber¹³ reports a case of a boy who became blind without ophthalmoscopic signs and with normal urine. The only assignable cause was latent scarlatina, and Leber seems to regard this peculiar case as somewhat analogous to the post-diphtheritic lesions in other nerves.

Occasionally accommodative asthenopia shows itself after scarlatina as it does after measles. It may persist for a long period of time even after the general health is completely re-established. Förster instances the case of a boy of 9 years who suffered from caries of both temporal bones and complete paralysis of both facial nerves. Both corneæ were destroyed in consequence of the lagophthalmus, and the unfortunate patient became blind as well as deaf. Fuchs, in his text book, states that suppurative choroiditis of metastatic origin may occur in scarlatina as in typhus, variola, etc. In most, if not in all of the recorded cases, suppurative otitis has preceded the choroiditis (or retinitis). Phillips¹⁴ has observed an œdema of the upper eyelid during scarlatina which also is apparently associated with suppurative otitis. The swelling was not white and doughy as in renal dropsy, but tense and livid, and in the cases recorded it was more marked before rupture of the membrana tympani, and increased afterwards if the discharge from the ear became less free. Phillips considers it probable that the affection is connected with thrombosis of the cavernous sinus.

As the result of this brief study of the more rare eye lesions that occur in scarlatina we are justified in arriving at the following conclusions:

First. In view of the fact that scarlet fever is one of the most common of the exanthemata, and that the majority of eye lesions occurring in connection with it are of a serious nature, involving not only loss of vision but in some cases life itself, greater attention should be given them than they generally receive.

Second. That if the operation on the cavernous sinus can be made without grave danger to the patient, and with the chance of lessening the mortality as claimed by the operators, such an operation is justifiable and should be performed.

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SOME RARE OPHTHALMIC CASES.*

By JAMES MOORES BALL, M.D.

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I. CONGENITAL ANOPHTHALMOS.

BABY C., female, was born in St. Louis on August 25, 1902. The mother was in labor twenty-four hours, and the delivery was instrumental. This was her first child. The mother was shocked by the death of a friend in January, and by a fire in February.

When one week old this child was examined by me at the request of the family physician, Dr. W. F. Kier. I found the babe entirely well formed with this exception—the eye-

balls were absent. The lacrimal glands were enlarged and tears appeared when the lids were held apart. The conjunctiva was of normal appearance and extent. Owing to the absence of the eyes there was a sunken condition of the lids, which is shown in the accompanying photograph. In front of each ear was a small, hard lump which was about one-half inch in diameter. Apparently these were inflammatory thickenings. They did not disappear. The little patient died in February, 1903, from inanition. Whether the masses were rudimentary eyeballs could not be determined, since a post-mortem examination was refused.



FIG. 1.—Congenital Anophthalmos.

Cases of congenital anophthalmos are of very infrequent occurrence. Von Hippel¹ has collected sixty-four cases of bilateral and twenty-three of unilateral congenital anophthalmos. In the twenty-three unilateral cases the other eye was normal in fifteen, four eyes were microphthalmic, one presented coloboma of the iris and chorioid, one showed a high degree of hypermetropia, and one presented an anomaly of the optic-nerve sheath.

II. DISCOLORATION OF THE CORNEA WITH BLOOD PIGMENT.

This is a rare and peculiar condition of the cornea—one which involves some nice points in diagnosis and which has been made the subject of study by such eminent observers as

Vossius,² Treacher Collins,³ J. B. Lawford,⁴ J. E. Weeks,⁵ and John Griffith⁶.

Mr. Wm. C., aged 37, native of Ireland, a blacksmith, was admitted to the St. Louis City Hospital in April, 1900. Two years before this time, while operating a punching machine, a piece of steel struck the right eye. The steel was a large piece and dropped from the eye. It is supposed that no part of it remained in the eye, although the history is indefinite. The eye was treated by a physician, and the patient states that one month after the injury an operation was made on this eye. He states that up to this time the injured eye was of the same color as its fellow, but immediately after the operation the right eye became of a reddish-brown color as at present. Before this time he could read large print with the right eye; immediately after the operation vision was reduced to light perception.

At the time of my examination of this case the entire cornea was of a brick-dust color. Oblique illumination showed the presence of wavy lines of vessel-like structures, which were probably distended lymph-channels.

In case of blood-staining of the cornea there is first a blood clot in the anterior chamber due usually to trauma or operation, although the hæmorrhage may have occurred spontaneously in old cases of retinal detachment. In nine of seventeen cases mentioned by Collins the tension was increased; in six others it was normal or minus. The phenomenon may occur at any age.

As regards diagnosis, it will be necessary to distinguish between hæmorrhage into the anterior chamber, forward dislocation of the lens, and blood-staining of the cornea. When the whole cornea is stained it cannot be distinguished from distension of the anterior chamber with blood, but if a peripheral clear zone is present the distinction can be made. When the central part of the cornea is of a rusty-brown color from blood-staining and the periphery is clear, the condition so much resembles that of an amber-colored lens dislocated forward that the best observers have been in doubt. It would seem that focal illumination ought to be of value in making the diagnosis.

As regards the nature of the pigment which is distributed

through the cornea, the micro-spectroscopic and chemic examinations of Collins show the discoloration is due mainly to crystals of haematoidin. with or without haemosiderin, which enter the cornea in solution in blood diffused through Descemet's membrane. After a period varying from two to many years the discoloration disappears, the periphery being the first to clear up. Treatment of this condition seems to be useless, although alteratives may be tried.

III. A CASE OF MELANOSARCOMA OF THE LOWER EYELID.

Miss C. D., of Jennings, Mo., aged 22, came to my clinic in February, 1903. When three weeks old she had a "gathering" in her left nose with discharge of pus. When the swelling subsided a small black spot remained just below the tendo oculi of L. E. After taking cold it was larger. Ever since she can remember there was epiphora of L. E. She came to me on Feb. 10th with pain and inflammation of inner canthus and adjacent skin tissues. A fluctuating oval tumor measuring 6x8 mm., was situated immediately below the left tendo oculi. This cyst I opened with a von Graefe cataract knife, evacuating about one drachm of yellowish fluid. The diagnosis of dermoid cyst connected with the upper part of the naso-lacrimal canal was made.

After evacuating the fluid the dark cyst walls remained. The cyst refilled until one week later the lower canaliculus was slit and a lacrimal probe was passed. This evacuated the fluid in the cyst. Ten days later I excised the growth. A microscopic examination, which was made by Dr. Carl Fisch, showed the mass to be a melanosarcoma.

¹ Die Missbildungen und angeborenen Fehler des Auges. Leipzig, 1900.

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MIDDLE EAR DISEASE IN TUBERCULOSIS.

BY ROBERT LEVY, M.D., DENVER, COLO.

The comprehensive title of my paper has been forced upon me by a careful review of the literature of tuberculosis as seen by the otologist as well as by my own personal experience. One has but to study the many short but pithy and varying descriptions of aural affections in tuberculosis beginning more than a quarter of a century ago, with Nelaton, Virchow, Toynbee, Zaufal, Politzer and many others, finally coming down to the most recent communications to the Otological Society of the United Kingdom, meeting of February 2, 1903, to be impressed with the chaotic state of our knowledge.

Upon all sides are we beset with doubtful questions of etiology, modes of infection, methods of diagnosis and plans of treatment. The tuberculous may be afflicted with any of the aural affections which attack the non-tuberculous. He may suffer an ordinary acute otitis media, suppurative or catarrhal, in which case the peculiar diathesis may modify or completely change the nature of the affection, or he may develop tuberculosis of the middle ear and adnexa as a primary affection or as a secondary complication of an already existing lesion elsewhere.

The usual modification of an acute otitis in a tubercular subject is manifested in the course the disease pursues. Whether suppurative or catarrhal, resolution takes place exceedingly slowly, the discharge persisting for a longer period or the deafness and tinnitus of the catarrhal variety continuing indefinitely. The latter variety rarely if ever becomes tubercular. In my practice such a condition has never been established to my satisfaction. Wingrave records one case of chronic non-suppurative otitis media in which tubercle bacilli were found.

Suppurative cases, however, may gradually assume the clinical appearance of the tubercular affection and tubercle bacilli may later be found in the discharge even though no sign of them was present in the beginning. These developments are of course only manifested in subjects suffering from tuberculous affections of other organs. It is a very doubtful question whether the tubercle bacilli in these instances, are present as manifestations of a tuberculous process. My own impression is that they are often found as accidental

constituents of a discharge laden with numerous and varied micro-organisms. My reason for this belief is that in the very careful bacteriological research made for me by Dr. Claude E. Cooper, tubercle bacilli were found with no regularity either as to frequency or numbers in the same class of cases or even in the same case.

The opportunities offered in the state from which I come, and which has been called a large sanatorium for tuberculosis, for the study of this disease in its development, its varied phases and its involvement of special organs are indeed numerous. Comparatively few cases of tuberculosis in children and relatively few instances of bone tuberculosis are met with, and these facts taken together with the infrequent occurrence of original cases in Colorado may account for the rarity of primary tuberculous otitis as well as for the moderate severity of the aural cases and the lack of extensive destruction of mastoid and petrous bones.

The majority of cases of primary tuberculosis of the ear seem to have occurred in children and the cases in which extensive caries and necrosis have occurred seem to have partaken of the character of bone tuberculosis.

In Colorado my experience has been that middle ear disease not uncommonly does develop in tuberculous patients but such cases are secondary to lesions of the respiratory organs, are chronic in their nature and pursue a slow and mild course. They often develop before the patient leaves the East and more than one of my records show that the ear complication manifests itself in the very incipency of the general disease, thus refuting the belief held by many that it is of late occurrence and an unfavorable prognostic sign. Moreover the ear trouble may appear at a time when the general condition is unusually good and when all systemic manifestations have been arrested. A case in point is the following: Miss May O., age 21, referred by Dr. Van Zant, February 6th. Patient has had lung trouble six months. Came to Colorado as soon as the diagnosis was confirmed. She began improving at once. The general symptoms were rapidly brought in abeyance, weight became above normal, temperature became normal, the patient feeling as well as ever in her life. Five months ago tinnitus developed in the right ear; three weeks ago deafness and slight discharge without pain were detected. Inspection revealed membrana tympani destroyed except for a small ring. There was very slight muco purulent discharge in the middle ear, the contents of which presented as red oedematous granulations.

Whether the tubercle bacilli be found in the discharge or not is in my opinion of minor importance. The process is either one of sim-

ple otitis modified by the specific vice or it is distinct tuberculous otitis. The bacillus tuberculosis may be present as an accident or as a causative factor, or it may be absent altogether and the clinical picture be identical with the most characteristic tuberculous process, as far as our present understanding goes.

It is quite easy to conceive how the tubercle bacillus may find its way into the middle ear either through the Eustachian tube from sputum, which being bacilli laden bathes the pharynx and nasopharynx, or how it finds the same destination through the lymph or blood currents, knowing as we do from many good authorities the frequency with which naso-pharyngeal and cervical lymphoid structures are affected with latent tuberculosis.

Many of my cases trace the origin of the ear trouble to the injudicious use of the nasal douche or the snuffing of cleansing solutions into the nose. For example, J. M. B., male, age 35, referred December 2d, 1902, by Dr. Zederbaum. Has had lung trouble ten months, has resided in Colorado six months. General condition of the patient is improving. One week ago while using the nasal douche patient felt a fullness in the left ear followed by autophony, tinnitus and slight discharge without pain. On examination found hearing for watch C/40, moderate voice 5 feet, bone conduction increased. Membrana tympani was perforated by a small round opening and bathed in scanty muco-purulent secretion containing tubercle bacilli.

The use of cleansing solutions in the nose and throat of tuberculous patients is to be commended and the douche is probably the most effective method of their application. Especial care should, however, be observed in instructing the patient in the manner of using the douche and the two points above all others to be insisted upon are first, to avoid "blowing" the nose for at least an hour afterwards and, second, to direct the stream into the more obstructed nostril.

Whether we consider this affection a distinct entity or a suppurative process upon which a tuberculous pathology is grafted, the clinical picture varies but little. The patient may or may not be aware of the immediate cause such as the douche or violent blowing of the nose. He feels for a variable length of time, a fullness as of a cloud in one ear, with occasional moist or dry rales. There is moderate deafness and tinnitus of varied degree and kind. As a rule there is no pain, perhaps only a sense of discomfort or possible slight twinge of earache. Very seldom a sharp pain is felt, which although unusual is not inconsistent with the disease. These symp-

toms may exist for a year or only a few hours before the more characteristic manifestations develop. These are found in the sudden perforation of the membrana tympani and the discharge of a small quantity of thin odorless liquid, usually watery or slightly milky in color and consistence. A moderate though constant constituent of this discharge is pus, the quantity of which determines its consistence.

With the exception of a very few acute cases the progress is exceedingly slow, but as a rule it follows a steady, destructive course. The small perforation, which is round and non-inflammatory, shows no inclination to repair but gradually enlarges until but little of the membrana is left. The middle ear is exposed with a pale pink œdematous appearance constantly bathed in the scanty characteristic secretion.

The hearing becomes steadily worse in many instances, but this is not constant except in the few cases attended with rapid and extensive bone destruction. It is not uncommon to find the deafness only moderate and stationary for an indefinite period. This discharge may at times appear arrested and remain so for many months, but as a rule this arrest is not permanent and under the least provocation such as slight cold or a mild relapse in some of the other tubercular processes the discharge will recur.

As to the development of miliary tuberculosis from this form of otitis as the focus of origin, I have never seen an instance.

In conclusion, I wish to make but a few suggestions in treatment. The object to be attained here, as in all forms of tuberculosis is to first cause an arrest of the disease. After this, functions may be restored in proportion to the amount of destruction that has taken place. Locally cleanliness is of prime importance. Cleanliness and the destruction of the tubercle bacilli may be accomplished by free irrigation with solutions of Formalin, in strengths varying from 1-5000 to 1-2000. The large perforations and the usual patency of the Eustachian tube enable one to freely irrigate the tympanic cavity. If the tube is not entirely open catheterization should be practiced. If granulations fill the tympanum they should be destroyed with a chemical caustic and completely shrunk by formalin or alcohol. If inflation and freeing the middle ear of granulations do not allow of through and through irrigation and thorough cleansing, the aural pump should be used and suction applied.

Of course all carious or necrosed tissue whether soft parts or bone must be removed, such means being employed as are necessary, from the mildest to the most heroic. Finally the greatest attention should

be paid to general treatment. The cardinal principles in the treatment of tuberculosis are a maximum of pure, fresh air, a maximum of sunshine, a maximum of rest, a maximum of most nourishing food. Climate plays a great role and wherever the air is purest and the sunshine most constant, there will the most be accomplished.

To summarize: First. Any of the usual aural affections may affect the tuberculous as well as the non-tuberculous.

Second. The usual modification of an acute otitis in a tubercular subject is manifested in the course the disease pursues.

Third. It is a doubtful question whether the bacillus tuberculosis is present as a distinctly etiological factor or as an accident.

Fourth. Comparatively few cases of tuberculosis in children and relatively few instances of bone tuberculosis are met with in Colorado.

Fifth. Clinical tuberculous otitis occurs with moderate frequency in Colorado, being secondary to lesions of the respiratory organs.

Sixth. Tuberculous otitis may develop when the general symptoms of tuberculosis have been arrested and the patients condition unusually good.

Seventh. Tubercle bacilli may find their way into the middle ear through the Eustachian tube, through the lymph channels or the blood currents.

Eighth. Unusual care must be exercised in the application of the nasal douche in tuberculous patients.

Ninth. The discharge may be arrested, but not permanently as a rule.

Tenth. It must be exceedingly rare for miliary tuberculosis to develop from an otitis as the focus of infection.

Eleventh. Through and through irrigation with tubercle bacilli destroying agents is of prime importance in the treatment.

Twelfth. General and climatic treatment must be conscientiously carried out.

DISCUSSION.

DR. J. A. STUCKY (Lexington, Ky.).—I am very glad to have heard the paper and to learn that there is very little difference between the tubercular otitis media and the ordinary suppurative trouble we meet with. I have had eleven cases wherein I suspected the discharge was kept up by the tubercle bacilli; I had bacteriologists make examinations but found none present, however. Recently I had a mastoid trouble as a result of otitis media. I operated the third time. Nine examinations had been made of the pus but we

found no bacilli until a week ago, and at the last operation we found tubercular disease of the bone. I agree, from my own experience, that very seldom do we find fully developed tuberculosis of the middle ear. I wish to ask about the use of the douche; I regard the post-nasal douche as dangerous in the hands of the patient, and I question the wisdom of allowing the average patient the privilege of using it. I doubt if any patient can do this effectually, and I wish to ask Dr. Levy what method he refers to when he says "use the douche frequently." He also advises the use of any of the solutions that will destroy the bacilli, and I would like to know what solution he would suggest that would destroy the bacilli and have anything left.

DR. JOHN J. KYLE (Indianapolis).—I agree with what has been said in regard to the great difficulty in finding the tubercle bacilli in the middle ear. With our somewhat limited clinical material, I know it is with us very difficult. Children suffer more frequently with secondary tuberculosis of the ear, rather than with primary. The great danger is in mixed infection. We find persons suffering with miliary tuberculosis of the lungs predisposed to a tuberculosis of the middle ear. There is, at least, a tendency to a catarrhal condition of the ear. This is followed by an active, acute purulent condition and a rupture of the drum which may be continued from year to year. You will find it difficult to demonstrate the tubercle bacilli in the middle ear. It is hard to isolate, though we look for it with great care. I do not believe it is always necessary to isolate the tubercle bacilli in order to make a diagnosis of tuberculosis. We can easily understand why the tubercle bacilli can get into the ear from the nose and throat. It is often found in persons of normal health, in the nose and throat, especially in those who are associated with tuberculous patients. These may migrate into the middle ear. If we had primary tuberculosis of the ear, we would have secondary tuberculosis in the lungs, because there is direct drainage through the Eustachian tube into the lungs. I believe that primary tuberculosis of the ear is a very rare disease indeed.

DR. E. L. SHURLY (Detroit Mich.).—I cannot add anything to what has been said. The reader of the paper has stated the facts in a very concise and intelligent manner. It is wholesome for some of the older members of the profession to listen to these facts, for a few years ago it was a crime for any man to consider that the tubercle bacilli were not alone responsible for all so-called tuberculous affections. The statements made by the author are, in my belief, true—as true as anything can be that comes within human

observation. I am especially impressed on account of some observations which I made on animals a few years ago. We had some monkeys treated by spraying into the nose solutions of sputum from advanced cases of tuberculosis. As long as this was kept up every day we could obtain and isolate tubercle bacilli. When the treatment was stopped for two weeks or so and the monkey isolated, the discharge from the nose would soon show no tubercle bacilli. Although we would put him among tuberculous monkeys again, we had to inject him again before we could find the bacilli. These monkeys were killed and an examination made of all the parts of the nose, but no tuberculous process could be found in the bony tissues or in the glands of the membranes. We examined the cheek pouches, the organs which correspond to our tonsils, and which are a most favorable place for culture media, and yet found no tuberculous process. The presence of the tubercle bacilli is probably largely a secondary event. The majority of these diseases are really of a mixed character.

DR. EDWIN PYNCHON (Chicago).—In Dr. Levy's paper he spoke of douches, and I understood he meant post-nasal douches, while Dr. Stucky evidently had reference to douches for the external auditory canal. I want to say one word regarding the benefit of douches: I have frequently used them in the external auditory canal in the treatment of inflammatory conditions, and only with the best results. I do not understand that Dr. Stucky wishes to denounce douches, but only the method of using them.

DR. JOS. BECK (Chicago).—I wish to make a few remarks in regard to some bacteriological experiments I have been making in the past year in the use of formalin in the ear, which I intend to bring out in my paper to-morrow. In answer to Dr. Stucky's question to-day. Heated formalin in various strengths was allowed to come in contact with culture media of tubercle-inoculated tubes, for various lengths of time, and cultures made from the same gave positive proof that the formalin from 10 to 15 per cent, heated, acted as a strong tubercle germicide. Forty per cent solution of heated formalin was allowed to pass into a middle ear infected with tuberculosis, and cultures made from the pus were innocuous. However, the ear became greatly irritated.

DR. JOHN A. L. BRADFIELD (La Crosse, Wis.).—I feel that what was said in regard to douches was due to the gentlemen not understanding each other. It is one thing for the patient to use the douche, and another thing altogether for the physician to do so.

DR. STUCKY.—The point I wish to make is that while I am not

opposed to the post-nasal douche in the hands of the physician, I am opposed to it in the hands of the patient, and I think we all have seen serious effects from its use, the solution being forced into the middle ear and there doing great damage. As to the use of the external auditory douche, I am not afraid of that, but I have never got a solution far enough into the middle ear to destroy the bacilli.

DR. LEVY (closing discussion).—I recognize the fact that some of my statements are at variance with our former teachings and with some of the good authorities of to-day, but I have placed on record the result of my own personal clinical experience, and I am ready to be convinced. The difficulties in the way of finding tubercle bacilli in the secretions of the middle ear are many. They frequently are there but are not obtained in the cultures we make. At other times the bacilli are there simply transiently, and during their sojourn in the ear, coming in contact with other micro-organisms, their appearance is so changed that they are not recognized. In the report by Wingrave in the *Journal of Laryngology, Rhinology and Otology* for March, he has shown that we have uncertain methods in examining these bacilli, and that there are so many different forms of tubercle bacilli that there is good reason to believe that oftentimes they are not detected.

The douche I referred to is the nasal douche. I understand from this an application of the douche from the anterior nares. If I had meant post-nasal douche, I would have so stated it. I believe this is the most dangerous manner in which to use the douche, unless very careful instructions are given.

As to syringing the ear, I spoke of a through and through method. Where the perforation is small it can hardly be done, but in this form of otitis, where we are dealing with a large perforation, the membrane being sometimes destroyed, I used the expression, "through and through" intentionally.

As to the use of formalin and other destroying agents, I presume the statement should be modified somewhat. I know that experiments are being constantly made in this line, and so far as I am aware at present the two agents which have the greatest power of destroying tubercle bacilli are carbolic acid and solutions of formalin. I am using these because they are tubercle destroying agents, but I did not say they are very destructive in the strength in which I use them. I dare not, of course, use the 40 per cent solution of formalin.

A DISCUSSION ON THE DIFFERENTIAL DIAGNOSIS AND THE TREATMENT OF OSTEO-SCLEROSIS OF THE MASTOID PROCESS.

BY OTTO J. STEIN, M.D., CHICAGO

The mastoid process of the temporal bone in the first few years of life is composed of fine cancellated bone tissue, which gradually undergoes absorption, giving place to the presence of a series of more or less well-formed air cells.¹

These cells communicate with one another and sprout, as it were, from the parent cell or antrum. They are lined with the same delicate and highly vascular mucous membrane as that found in the tympanum and antrum.

The mucous membrane lining the mastoid cells plays a double role, in that it is mucous membrane to the cell cavities, and periosteal covering to the bone. The cells receive their secretion from the membrane, the bone, its nourishment.

A chronic congestion of the membrane results in a low grade of inflammation that tends, on the one hand, either to thickening or to pus formation, and, on the other hand, to an osteitis with a resulting hyperostosis, caused by the hypernutrition, or pus formation as the result of caries or necrosis.

These conditions, of course, may co-exist or occur independent of one another. The hyperostosis may exist as an idiopathic disease, the result of a previous inflammatory condition of the tympanum or antrum, but developing itself after the latter had subsided; and, on the other hand, it may develop as an accompaniment to an active morbid condition within the tympanum or antrum, or both.

In the hyperostosis we have the formation of new bone cells from the periosteum and also from the medullary spaces. This proliferative process may continue so that all of the cells may be obliterated, the new bone tissue finally becoming so compact and hardened as to merit the name of "ivory" or "ebony-like." This process is known as osteosclerosis or eburnification of the mastoid bone.

It is very easy for us to trace the development of our knowledge on this subject, because it is a knowledge of but comparatively few years.

The first recorded reference to the subject that I could find is in

the early writings of Schwartze and Politzer. Vague, incomprehensive, and perhaps doubtful as they may have seemed, they nevertheless directed the inquiring and progressive otologic mind in the direction that has developed into a knowledge that to-day gives to the subject a distinct and individual place in the pathology of mastoid disease. What the subject still lacks, though, is a clinical picture that will awaken in the mind of the otologist the necessity of differentiation between conditions productive of similar symptoms, which, thoroughly understood, gives to him the requisite conviction to apply a remedy potent with decided and prompt relief.

Schwartz² says: "Sclerosis is a frequent sequence of chronic purulent inflammation of the middle ear, the cells gradually contracting and finally disappearing."

In the report of the American Otological Society of 1870, Dr. C. R. Agnew,¹⁸ of New York, probably makes the first recorded reference to this condition, in the following words: "Caries is not the invariable and immediate result of mastoid-cell disease, but sometimes there may be, instead, an osteitis, with hyperplasia of the bone, filling a few or all of the cells."

From this time on several investigators followed up the subject very carefully, and in 1873 Buck,³ in an article on mastoid disease, referred to the condition under the head of "Hyperostosis of the Mastoid Process."

Shortly following upon this time, 1876, J. Orne Green,⁴ of Boston, Mass., as is shown in the report of the International Otological Society of 1876, and in the Transactions of the American Otological Society of 1880, attempted to diagnose a specific mastoid disease known as Hyperostosis of the Mastoid Process; and three years later Dr. Arthur Hartmann,⁵ of Berlin, published in the Archives of Otology, 1879, a paper in which he sets forth a statement recognizing that an idiopathic disease of the mastoid process may exist as an osteosclerosis with definite symptoms.

In all the four cases reported by Green, in 1876, in connection with his article on Hyperostosis of the Mastoid, there was present a chronic purulent inflammation of the tympanum, associated with the hyperostosis of the mastoid, and sudden acute symptoms arising, operations on the mastoid were carried out, with the result that nothing but a hyperostosed condition was found, although complete relief from pain was brought about by the operations.

In three cases reported by Buck,⁶ in 1883, all were associated with running ears in their early history. One case was lost sight of, the others were operated upon solely for the mastoid pain, the discharge

having stopped for years, and complete relief was afforded after the patient had suffered for several months.

A most excellent exposition of the condition is given by J. A. Lippincott,⁷ of Pittsburg, Pa., in his report of "A Case of Mastoiditis Interna Chronica with Sclerosis," before the seventeenth annual meeting of the American Otological Society, 1884. In this case, like the two cases reported by myself,⁸ the chief symptom was pain, without any marked evidence of existing middle-ear disease that would otherwise demand relief. This case, like my own, made an excellent recovery after trephining the mastoid process.

From the foregoing remarks it will be seen that there are two varieties of this condition: one where the sclerosis is associated with a suppurating process within the tympanum or antrum, and the other without any associated suppuration. It is with the latter that I wish to deal particularly. In this latter variety we may include those cases that may have a history of a former suppuration, as well as those in which no such history or existing evidence can be had.

In the simple or uncomplicated variety of mastoid sclerosis the symptoms are few in number. Hence it becomes a matter more of differentiation between disorders with similar symptoms. No doubt many cases of hardening of the mastoid process exist wherein the patient complains but little or perhaps not at all. But in cases where pain is complained of, there is little else, aside from this symptom, that remains characteristic of the disorder.

Hence pain is the preponderating symptom present, and one from which the patient seeks relief. The subject, therefore, resolves itself into a discussion of the varieties of ear pains and their characteristics.

A chronic pain in or around the ear may be studied under four heads:

First, otalgia, associated with an internal or middle-ear or antrum trouble.

Second, hysteria, neurasthenia, and malingering.

Third, neuralgia from other causes than ear troubles.

Fourth, osteosclerosis of the mastoid process.

Otalgia from Associated Ear Troubles.—Pain, as a result of trouble with the external, middle, or internal ear, reveals its true character mainly in its association with such disturbances. As, for instance, a foreign body or a neoplasm within the ear canal is seen upon careful inspection, and with the removal of the same the pain disappears. Pus, granulation tissue, or cholesteatoma in the middle ear or antrum may be demonstrated either by ocular examination, the use of the probe, microscopic examination of the washings of the ear.

and by other well-known methods. In all these conditions the character and duration of the pain differ from that of osteosclerosis, in that it seldom reaches the acute exacerbation; nor is it so persistent or prolonged, and, as a rule, it is amenable to some of the ordinary measures of treatment.

Hysteria and Neurasthenia.—The chronic pains of the malingerer, the hysteric, or neurasthenic may at times be more difficult to differentiate. In all such cases we must be broad-minded and far-seeing enough to take into consideration the entire domain of medicine. For instance, in order to eliminate the possibility of a neurasthenic condition, we must ask ourselves whether our patient is suffering from the results of an exhausted state of the general nervous system. Is he in a depressed mental state, associated with a worn-out and tired feeling, lacking ambition during the day and wakeful at night? If so, further inquiry will reveal the fact that he has been working under pressure or at a high tension. Long, hard days, and perhaps nights of exacting or tedious duties, coupled with a disregard for proper eating and necessary exercise, establish in the mind of the physician the true nature of the malady.

On the other hand, should our study of the case bring to light some of the following symptoms, we may reasonably suppose that we are dealing with a condition of hysteria; an inclination to complain of the physical or mental state; a tendency to exaggerate existing symptoms; given to extreme expressions of emotions; great imagination; subject to disorders of sensations, like globus hystericus, tinnitus aurium, epigastric pains, headaches, eructations, tympanites, chilly sensations with yawning and stretching, diuresis, muscular twitchings, cramps, convulsive attacks, neuralgia, hyperæsthesia, anæsthesia, analgesia, aphonia, functional paralysis, cough, retention or suppression of urine, rigidity of neck, tenderness of nape of neck, etc.

So much for generalities. Now let us look more particularly at the aural symptoms. In the earache complained of by the hysteric the pain more frequently is located in the auditory canal and in front of the ear. Besides, pain is referred to the temple. The mental state, as well as the condition of the general health, influences the severity of the pain. That is to say, when the patient is much depressed he is sure to complain most; but when his spirits are at their best he complains none at all, or but little, and then the location of his complaint is but ill-defined, or it is located in an entirely different place.

One of the characteristic things about hysterical affections of the

ear, as Gradenigo,⁹ Politzer-Bruhl,¹⁰ and others have pointed out, is that there is an association of sensory disturbances about the parts. While the patient may complain very severely of pain within the ear and about the mastoid region, careful examination frequently discloses anæsthetic or hyperæsthetic areas about the pinna or auditory canal, as well as in other parts of the body. Moreover, the patient may complain of paræsthetic sensations somewhere within or about the ear, like a sense of formication within the canal, or as if there were something animate within, and again as if there were a slight discharge. Some cases present neurotic disturbances of the eighth nerve, manifesting themselves either as a hyperacusis or paracusis, tinnitus aurium, periods of momentary deafness, nausea, and vertigo. In other cases there may be present transitory disturbances of the motor nerves of the face, like twitching of the muscles or even a paralysis. Often the opposite ear becomes involved without any objective symptoms.

Voss,¹¹ in discussing the subject of ear disease and hysteria, says the diagnosis of hysteria must in the first place be supported by heredity; the disturbances of cutaneous sensations are important; the anæsthesia varies as to time and place; the tuning-fork generally is not heard by bone conduction on the affected side; and finally, the condition is generally found in women between the ages of twenty and thirty years.

Lannois and Chevanne,¹² from their large experience and the great number of cases treated, advise the employment of suggestion in the treatment of hysterical mastoid cases. Their good results in these cases give to us an additional means of differentiating between an imaginary and a real affection.

Jendressik¹³ describes a number of cases of neurasthenic neuralgia and says the subjects always exhibit the stigmata of neurasthenia, and that a hereditary predisposition can be almost always discovered. There are none of the objective phenomena which accompany genuine neuralgia. The patients are able to keep their mouths and teeth clean, and like to do so when the neuralgia is in the face, while in genuine neuralgia they shrink from the slightest contact with the parts.

Neuralgia from Other Causes.—Under the heading of neuralgias we have a long list of disorders that may give rise to ear pain. The pain due to chlorosis, anæmia, malaria, and influenza may, aside from other symptoms diagnostic of their nature, be differentiated by characteristic conditions of the blood as found in these representative disorders.

It is very proper at this point to emphasize the importance of securing additional aid to clear up the diagnosis, by the examination of the blood and urine. The examination of the blood has become almost a universal practice among surgeons of to-day, and particularly as to its significance in acute suppurative processes.¹⁶ It is interesting to note in this connection a case reported by M. D. Lederman,¹⁴ which clinically simulated mastoiditis, but on exploring the mastoid process nothing was found, the case afterward proving to be one of malaria.

In case of syphilis, the history of an infection and the knowledge that the pain is not so persistent but characteristically severe at night, are usually sufficient upon which to form an opinion.

In rheumatism, the involvement of other parts of the body, the marked periods of remission from pain, and the aid received from urinalysis suggest the nature of the disorder.

Ear pains as a reflex expression of a disordered condition are not uncommon. Instances of such disorders are seen as the result of diseased teeth, gums, and the tongue. Korner,¹⁵ in discussing neuralgia of the ear, refers to a case of tympanic neuralgia due to an abscess in the tongue. The pain was increased when pressure was exercised on the hyoid bone. He has repeatedly observed the increase of pain in the ear of tympanic neuralgia from carious teeth, on pressure in the hyoid region, and considers this a diagnostic symptom.

Other reflex pains may arise from nasal or laryngeal disturbances. Pain in the ear has been reported as a result of caries of the vertebræ, and similar reflexes have originated from the brain and sexual organs.

The pain of neuralgia shoots in the direction of a nerve trunk and its branches. In cervico-occipital neuralgia the painful and tender points are situated as follows: between the mastoid processes and the cervical vertebræ; on the parietal side of the head; on the mastoid process; in the concha, and it may also be manifested in the temple and the ear canal. A point well borne in mind is that cold or heat, when applied to the affected nerve or its branch, frequently aggravates the pain.

One will at times observe neuralgia in a case of debility following a siege of some acute affection or some chronic disease.

Osteosclerosis of the Mastoid Process.—The symptoms of osteosclerosis are mainly centralized into that of pain. In the uncomplicated variety of this disease the almost negative results of our examination strengthen our diagnosis. On inspection of the drum

membrane, most likely nothing of any note will be seen, although it may show the usual changes that are present as a result of a former otitis media catarrhalis or suppurativa. The retro-auricular region seldom shows anything pointing definitely to the involvement of the interior. In some cases, particularly when accompanied by a chronic suppuration of the middle ear, or in cases of cholesteatoma and also in the formative stage of the simple variety, we may discover a slight redness of the overlying skin and a light degree of œdema. Firm pressure over this area will discover a spot, usually located on a line with the meatus, that elicits a sharp sensation of pain. The tenderness is not limited necessarily to this region, but at times it may extend over quite a large area. But what is particularly worthy of note is that the auricle, integument lining the auditory canal, the region in front of the tragus and just beneath the lobe of the ear, are not at all sensitive to the touch.

The pain complained of by the patient is almost always continuous and extends over a long period, from days into weeks, and weeks into months, with only remissions, but seldom intermissions; although at times, by the mere fact of its long continuance, the patient may become so accustomed to the milder stages of the pain as to be apparently unconscious of its presence.

The pain is accompanied at times by sudden exacerbations of an acute throbbing or boring character, deeply seated in the ear and mastoid process. In some instances it may radiate up to the side of the head and down into the neck and again back to the occiput, but, as a rule, it is not associated with pain in or anterior to the meatus.

Deafness may be complained of according to the amount of involvement of the conductive or perceptive apparatus.

Usually there is an absence of tinnitus and vertigo.

A slight rise of temperature may be noted in the early stage of the disease when the new bone tissue is forming, but later, during the hardening period, it is never present.

Age does not seem to bear any causative relationship to the disease.

The employment of auscultation, percussion, or transillumination has as yet proven of no convincing value to me in the diagnosis.

One final word now as to the possibility of error in determining between a purely neurotic disorder and one of osteosclerosis, and that is, in the proper individual, who is susceptible and impressionable, the persistent, nagging, torturing, and often agonizing pains incident to a case of osteosclerosis, may soon bring forth the latent qualities of a full-fledged hysteria, just as it brings forth the demand for powerful sedatives and analgesics to relieve him of his suffering, until

finally he finds himself a helpless, miserable, and pitiful subject of the drug habit.

Treatment.—In considering the treatment of this condition, we may divide the subject into the medical and the surgical.

Under the medical treatment we will refer to all such measures employed other than that of operative.

Remedies of this class, to be of any particular value, should be used in the early stage of the disease—that is, where there is great vascularity as a result of the new bone-forming process. At such a time the various rubefacients or epispastics may be tried, such as mustard, capsicum, cantharides, ammonia, camphor, and turpentine. The methods of “firing” and acupuncture are little used nowadays, but in their stead we may find the employment of the X-rays and mechanical vibrations. The leech, dry cups, and galvanism are remedies familiar to all.

The surgical treatment is the important part of the therapeutics of the disease. Notwithstanding the advice and practice of such authorities as Politzer,²³ Hartmann, and Knapp, one is amazed at the apparent apathy, part ignorance and also prejudice, existing relative to the employment of operative measures in this particular condition.

As far back as 1875, Professor Gosselin¹⁷ read a paper before the Paris Academy of Sciences, entitled “Osteo-Neuralgia of the Long Bones,” in which he advises trephining in order to relieve the pain. That this same view is held to be applicable in cases of osteo-neuralgia of the mastoid is seen by reading the opinions of men like Politzer, Green, Buck, Hartmann, Schwartze, Knapp, and others.

Buck says: “We should not hesitate to perforate the bone,” in order to bring about relief in these conditions.

Schwartze,¹⁹ the pioneer in mastoid surgery, the man to whose personal efforts through research and indomitable courage aural surgery owes a great deal of what is good and beneficial to-day, advocated and practiced operating on the mastoid in cases of intense and uncontrollable pain of the mastoid process.

Hartmann⁵ says: “Practice proves that the symptoms of violent pains, in connection with idiopathic sclerosis, can be relieved by opening the mastoid process,”

Sattler²⁰ cites numerous cases illustrating the condition under discussion, which upon operation gave permanent relief.

Herman Knapp²¹ has repeatedly operated for osteosclerosis of the mastoid process, with success as to the relief of pain in all cases.

Trephining the skull is practiced by many surgeons for the solitary symptom of headache. Siegel²² cites such a case and reports finding

eleven similar interventions on record. All cases are reported cured by the operation.

As I have already stated in a previous paper on this subject, after having made your diagnosis and exhausted the simpler means of relief, an operation of opening into the process is the only rational, positive, and successful means of putting an end to the patient's suffering.

Where we decide to operate in a case associated with hysterical stigmata, it is important not to burden the patient's mind with any doubtful prognosis, but, on the contrary, carry conviction with our words by predicting a most hopeful outcome.

In operating, our object is to relieve bone tension, and with this in mind we should remove as large a core of bone as possible, making the superficial circumference of the opening of large diameter, and the edges and surface of the cavity clean and smooth.

In the uncomplicated variety of this disease, it may not be necessary to penetrate into the antrum. In fact, the rule is not to.

In conclusion, allow me to urge upon you the recognition of a broader field for surgical intervention in affections of the temporal bone. The modern surgery of the mastoid process has given to us the ways and means whereby serious symptoms and fatal results may be thwarted, and the revelations made to-day by such procedure explain the lethal cases of former years. Although the condition of sclerosis is not, as a rule, associated with the possibilities of a fatal issue, it very often makes itself manifest by such uncontrollable pain that it leads the patient a life of intolerable suffering, which evokes within him such a strong desire for relief that the drug habit is soon acquired. In the face of such a condition or such prospects, can we otologists of to-day remain unmindful of the mental and physical deterioration that results from such state of affairs, and with a remedy so potent and powerful in our hands refuse to be moved by the sense of what is our proper and legitimate duty?

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DISCUSSION.

DR. JOS. BECK (Chicago).—This subject of Dr. Stein's has interested me very much, because I have followed it since he has presented it at the Chicago Medical Society, where it created such an unfavorable impression. I am going to operate on a case of that kind, and I would ask your indulgence in giving a brief history of the case. Bishop operated this case for mastoid disease on the same principle that Stein has mentioned. Three years ago he opened the mastoid in a woman 45 years of age; slight suppuration at one time in both ears. The suppuration ceased, but the pain did not, either on the side he operated or the other. The pain has been excruciating but it is not continuous. It lasts sometimes as long as a month. Strong narcotics have to be employed, and the patient is a physical wreck as a consequence. I believe that not until these mastoid cases are operated on and bone examined microscopically can we speak confidently of osteo-sclerosis, but it will be interesting to me to watch this case and see if the pain will stop after the operation. I will hope to be able to present something in addition to this from the pathological side.

DR. BARNHILL.—I have seen several cases of osteo-sclerosis of the mastoid process, and have operated on a few with good results. I should hesitate to operate, however, on cases with no other symptoms than pain over the mastoid, and an old scar in the membrani tympani. My cases have all had chronic discharge from the ear, and I have looked upon the length of time the ear has discharged as being of great diagnostic importance as to the amount of eburnation in the

process. I recently saw a case in which I believe a mistaken diagnosis had been made, and a part of the mastoid process chiseled away in the belief that there was sclerosis. In this case the membrana tympani was perfectly normal, and so was the hearing. There was chondritis of the external auditory meatus, and infiltration of the cervical glands; also numerous decayed teeth and pyorrhoea alveolaris, which oral conditions had much to do with the pain of which the patient complained. I should like particularly to have Dr. Stein state whether or not in his opinion osteo-sclerosis takes place to any considerable extent long after the suppurative process which caused it has ceased to be active, and after the perforated membrana tympani has healed.

DR. W. L. DAYTON (Lincoln, Neb.).—If I understood Dr. Stein correctly, he mentioned that in operating it was not always necessary to open up the antrum. In some of these sclerosed mastoids, we find that the antrum is but a small excavation, but the necessity of opening up to the antrum where pain was a symptom was illustrated in a case operated by Dr. Davie of the Illinois Eye and Ear Infirmary about a year ago. As I remember the history, there was absolutely a normal drum head. He opened the antrum and found it full of a purulent secretion, or rather, a muco-catarrhal secretion. Another case was that of a young man who had been stealing a ride on a freight train and was thrown from the train by a brakeman ten years ago, striking his head in the fall. Since that time he had had a chronic purulent otitis media. He came to me in almost a state of collapse. There were at that time no symptoms—such as slow pulse, high temperature—of abscess of the brain, but from the mental state I feared it. I determined to operate on the mastoid and then trephine over the temporal lobe. I opened up the sinus, in which I found granular tissue and removed it from the dura; I then opened nearly over the tip of the mastoid to the mastoid squamous suture and found only a slight depression where the antrum should be. The anesthetist positively refused to give the chloroform further (we had made a mistake in giving chloroform) and I was obliged to stop the operation and put the patient to bed, thinking that the next morning I would open the skull. But the next morning he was too far gone and he died that afternoon. At the post mortem it was shown that he had an abscess in the temporal lobe, and no cells in the mastoid process larger than pin heads.

DR. W. R. MURRAY (Minneapolis, Minn.).—This paper has been very interesting to me. My experience with these cases has been limited, but six or seven years ago, at the Eye and Ear Infirmary in

Chicago, a case was kept under observation for a considerable time before a definite diagnosis was made. The patient was a woman, somewhat neurotic; her tympanum was normal. She complained of a constant pain over the mastoid, neuralgic in character. The operation was finally done and the mastoid was found to be sclerotic, of intense hardness like ivory. The case was lost sight of after the wound had healed, but up to that time there had been no recurrence of the mastoid pain.

DR. STEIN (closing discussion).—I wish to disabuse your minds of the idea that one can diagnose this condition from any one or from any few symptoms in all cases. It is very difficult to make a positive statement as to the condition of osteo-sclerosis; it is to be made by exclusion. There is nothing tangible in literature upon the symptomatology of this disease that I can find. I have read some excellent articles upon the subject, but nothing from which we can positively diagnose this condition. I do not pretend to present an array of symptoms from which you are going to be able to diagnose this condition with a certainty at all times, but simply have given you what I have read and also what I have seen and learned from cases operated upon myself. You may take whatever of worth it may contain. The symptom of pain is the main thing and the one for which the patient applies for aid. It is usually the only symptom.

Dr. Barnhill in his question has emphasized the very point I wished to make. He said he would not diagnose such a condition from a simple scar of the drum membrane associated with the pain. I would not either. But in this uncomplicated variety you may find evidence of a former ear trouble in the scar or perforated drum membrane. This brings us down to the causation of the disease; is it primary or secondary? I could not answer the question now. Very likely it is a secondary process. Some claim it is an idiopathic disease. That is not the question I am bringing up.

In regard to opening the antrum, that has to depend upon the case. Probably in the majority of cases I would open the antrum, but if I had a case without a history or symptoms of mastoid trouble excepting this pain, I rather doubt if I would enter the antrum. In one case I remember distinctly of going as far as I dared and finding no antrum. There was no sign of a cell, and I took off the whole process. I do not know if I have made myself clear, but I hope I have.

REMARKS ON THE ETIOLOGY OF HYPERTROPHIC RHINITIS.

BY E. L. SHURLY, M.D., DETROIT, MICH.

In discussing the subject of hypertrophic rhinitis, it would seem impossible, as you know, to avoid all speculative philosophy, for notwithstanding the great practical progress in rhinology which has been made during the last 25 years, many of the out-cropping problems are still sub-judici. This condition probably arises from the fact that we have no natural anatomic or physiologic standard applicable to civilized man—because he is, so to speak, an artificial animal.

The commonly found abnormal hyperplasia of the turbinate tissues, regarded from a critical standpoint, seems to be one of the most senseless irregularities of nature. Rarely in any other place in the body is nature's conservatism so conspicuously absent. Reflecting upon general lines, it appears really absurd that passages like the nasal—so important for the maintenance of health and good respiration—should be so often, and I might say, so unnecessarily deformed in so many human beings of all ages. Therefore, in approaching the topic of its etiology, many factors must necessarily attract our attention.

In the first place we may fairly assume that one of the basic predisposing causes, at least of nasal turbinate hypertrophy or deformity in civilized man, may be attributed to the evolutionary or devolutionary processes which the structures have been undergoing through many generations, perhaps, in reaching the present condition (size and quality) of the cerebrum and face. This assumption, no doubt, might be seriously questioned if there were an absence of any positive demonstration to show that the head and face of the Caucasian at birth varies at the present time very much from that of centuries before. So far, however, as we can rely upon archeological data, it would seem that the relative proportion (small face and large calvarium) of the face and calvarium at birth has undergone through all of these ages a slow but certain change, so that now the facial parts of the Caucasian at birth are strikingly diminutive and compressed as compared to the other parts of the body. This theory, if we can so designate it, is further reinforced by

accumulating evidence showing a relatively greater difficulty in the process of parturition than occurred toward the primitive stages of the Caucasians racial existence. Furthermore, there is a strong probability (although investigation has not been made as fully as it ought to have been) that the Malay and Mongolian races, for instance, do not vary so universally as does the Caucasian from a purely natural type—so that neither the proportions of the foetal head are as large at full term, nor the maternal pelvis as small proportionally as those of the modern Caucasian.

It would, therefore, seem probable that possibly pre-natal, as well as post-natal, processes of development serve in a great measure as predisposing causes of the abnormal condition in question, because you are all aware of the fact that in civilized Caucasians more than 80 per cent bear the marks or unsymmetrical or abnormal developmental conditions of the upper respiratory passages. However, we have no time to go further into these interesting topics—the hereditary transmission of acquired proclivities, or the laws of catagenesis, further than to call attention again to the fact of the gradual and progressive declination of the Caucasian race from its aboriginal physiologic type,—especially in respect to the osseous frame work of the face. The particular devolutionary process by which the nasal region fails of development need not detain us, because this process has been formulated theoretically by different writers several times a year during the last quarter of a century. Although interesting, it is a subject which we leave for the embryologists of the future, in the hope that they may discover the several steps of this early operation of nature. Suffice it to say, that departures from aboriginal conditions may and undoubtedly do in many instances give rise to the hypertrophic rhinitis so often occurring in early life.

One of the particular causes of the disease as has been often stated by observers is a neglected or overlooked ethmoiditis, or osteitis of the suppurative type, or some latent infectious disease of the accessory sinuses—either the frontal, sphenoidal or maxillary. Whether such conditions in infancy are connected in any accidental way with suppressed post-natal development of the facial bones is still an unsettled question, or whether such conditions are always the result of a primary sepsis is yet unsettled. Lastly the question of syphilitic taint in such infants has been brought forward from time to time as a common cause.

A wide range of inquiry among the old and well-settled general practitioners, to say nothing of statistical inquiries in that regard,

would seem to indicate that the number of persistent chronic, so-called nasal catarrhs of whatever description met with in infants, is very small, while on the contrary in childhood it is very large.

Hence, it seems to me that we should firmly plant our belief upon this ground—namely, that the upper nasal passages in the Caucasian infant are only abnormally normal, and consequently a menace to the individual; that through this lack of typical structural integrity, and a consequent devolutionary degeneration which may be ascribed to the law of disuse (such as the degeneration of the sense of smell) a positive tendency to pathologic conditions is created. This being the case, it is easy to see how the lining membranes of these parts—in consequence of their highly vascular and neurotic supplies, and the limited space afforded naturally for the growth and development of all the integral elements of their composition, may become—as life advances and the various acute diseases of childhood are acquired—the seat of incessant histologic change.

Next, taking into consideration the artificial environment and mode of life of the modern Caucasian—(an artificial animal) we can readily understand how easily this may operate upon the already abnormal structure or other physical defects, or physical tendencies, which we have just been calling attention to. With the individual thus poised—so to speak—between a physiologic and pathologic line, it is obvious what results must follow the exciting causes which exist everywhere about in the form of diseases peculiar to childhood, as well as a generally pernicious environment. I would not be understood as stating that every case of hypertrophic rhinitis commences in childhood, for many of them, we know, have begun during youth and adolescence. Many of them come on later in life, undoubtedly from some peculiar alteration in the function of the sensory nerves or ganglia (such as probably occurs in hay fever, or by the passages of motor impulses over sensory filaments, and vice versa); while many cases undoubtedly owe their origin to either untoward occupations or habits.

It seems to me among the most potent causes of hypertrophic rhinitis,—next to the lack of proper development of the nasal passages, naso-pharynx and accessory sinuses,—are attacks of measles, diphtheria, scarlet fever or the exanthemata generally. It is really very rare for any child to go through a course of one of these diseases without being left with some hyperplasia of the glands or vessels connected with the mucous membrane of the upper air passages—"the lymphoid ring." This is a matter of such common observation that it would seem beyond dispute. I speak now in a

relative sense, because we have no absolute standard of size or condition for any of the glandulæ of the lymphoid ring. Indeed, the question of a diseased state hinges upon whether (a) there is too much secretion, (b) altered secretion, or (c) not space enough to contain the glands as they exist. Another great source of hypertrophic rhinitis which seems very obvious consists in the unhealthy mode of life which is pursued by the civilized Caucasian. Among the well-to-do class," the bodily dressing is either too much for indoors or too little for outdoors. Houses are too hot and illy ventilated; the food is too much, of too great variety, and too complex in its mixture, to say nothing of the use of too much alcohol. With the vast array of "fake" foods, and spreading indulgence in pernicious habits, the wonder is that more persons are not unhealthy. With the poorer class, there are similar defects of living—namely, too much clothing in the house; too much artificial heat; illy ventilated rooms; not enough cleanliness; too much alcohol used; and too much poor cooking. Keeping in view the function of the turbinate bodies—which is to become vascular and swell in order to protect the lower air passages from exposure to very cold air, by the radiation of heat to the incoming stream of air while at the same time mechanically diminishing the space for the limitation of the passage of the same, when necessary; too much stress cannot be laid upon the bad effects of exposure of the nasal passages to the sudden extremes of cold and heat alternately. Yet how many persons are thus exposing themselves during the winter season—going out from a dry-air of a temperature somewhere about 80 degrees into air at a temperature of from 40 degrees to below zero, thus straining the equalizing functions of both skin and nasal mucous membrane to the utmost. Frequent acts of this sort are likely, of course, to produce hypertrophic rhinitis, as well as other naso-pharyngeal abnormalities.

I apprehend that soon a newer etiologic factor will arise in the prevalent consumption of fake foods and nostrums. Through these foods and things, as the quantity of their consumption grows through the medium of that all powerful evil of dishonest advertising, there certainly will be a reactionary effect upon the glandular respiratory system originating in the digestive system. For we all know that abuses to the metabolic functions though expressed in one apparatus will effect others through the medium of the connecting branches of the sympathetic nervous system. In conclusion, it seems to me that this condition known as hypertrophic rhinitis—excepting for a few cases which may be directly traced to occupation or exposure—

marks a process of physical alteration which has pursued our race in a ratio proportionate to our departure from the natural life of our aboriginal ancestors. It is therefore one of the conditions that will steadily prevail more particularly, of course, in certain climates than in others, and its defects and consequences will have to be met by medical and surgical art. We can scarcely hope for its eradication under the present conditions of human social affairs; but its amelioration can be gained by the careful supervision of the medical profession in guarding individuals and communities from the consequences of infectious diseases and unphysiological social life. In short, the Caucasian child, born with a preter-natural cerebrum and preter-natural face, reared to an artificial life, deprived of a full opportunity to become adjusted to the natural climatic environments, and doomed to an artificial life of indoor or abnormal activity throughout youth, adolescence, manhood, and womanhood, is destined to be the selective field of strife between those two cardinal evolutionary forces of nature—namely, constructive or organizing metamorphosis, and destructive or disorganizing metamorphosis.

DISCUSSION.

DR. G. V. WOOLLEN (Indianapolis).—Dr. Shurly has shown very clearly that he has been with his subject a good while, and that he has delved in to the primary conditions, as we are not apt to do in our earlier experiences. The doctor has been at this long enough to know that it is not to be explained in a word. The philosophy underlying his remarks is profound and instructive because he goes back to pre-natal conditions. Some of you will remember that I read a paper before the American Laryngologic Society at Chicago on "Taking Cold." This subject is so nearly related to taking cold, or to the conditions we indicate by the phrase "taking cold" that it makes them one and the same, because you cannot have the hypertrophic condition without having the so-called cold or catarrhal condition. When you deal with this condition as Woakes does in his work on nasal catarrh, you go into the subject with which the doctor deals. He abuses alcohol greatly because he can see the ill effects of it which he confines to the Caucasian race. I have been racking my brain in reference to my own experience with the other races to discover whether or not he is justified in what he says, and I believe he is more than half way right. Our Ethiopian friends do not trouble us much in this way. I had not thought of the facial contour and brain as he suggests. When we come to the later manifestations, such as those we find in children, we are on familiar

ground and have a better chance to study. Since my earliest teachings, I have insisted that this was a disease of childhood. I believe that none of you have met with a case of hypertrophic disease of the nose but that had developed during child life. I believe it to be, as I stated before, pre-eminently a disease of childhood. The first evidence will be on the anterior tip of the turbinal and a little red spot will show its location. I have been accustomed to teach that this disease has an early inception (without going back to the pre-natal condition as the doctor has to-day)—that it begins first because of the physiological and second because of the anatomical construction of the nose. I have used as my first illustration to my students, the gills of the fish. As in these we have a physiological hyperæsthesia as compared with other parts of the body, and we have also a physiological hyperæmia of the nose as compared with other portions of the body. We must have these in order that the nose may perform the functions for which it was designed. Again, the nose is pre-eminently the seat of traumatism. In our childhood, our first efforts to roll over land us on our noses, and see the effect of these early traumatisms in the deflected septum, etc. As to our rooms, I have long felt that more harm was done by the overheating of rooms than by the underheating of them. We do not “take cold” as much as we “take heat,” and then there is the dust. In places where this prevails intra-nasal hypertrophy is rife, and it is probably as frequent in Indiana as anywhere. The vicissitudes of the temperature, etc., are also important factors.

I enjoyed the doctor's remarks immensely and it is one of the greatest subjects that could possibly be brought before the Academy.

DR. H. STOWE GARLICK (Cincinnati, Ohio).—I was struck with the idea—new to me—that as a process of evolution we should have this hypertrophic rhinitis. One of the points not clear, I think, is that it is not a disease of childhood except as a result of certain outside causes which the doctor mentioned, and if that is so, how can he attribute the process to evolution? In the comparison of the Caucasian race to the Malays, for instance; the latter people are not exposed, as the civilized are, to the extreme changes of temperature, the massing together in colonies, and the dietetic influences to which we are all subjected, and it seems to me that before we can accept this theory it will require more careful investigation. The pre-natal influences of heredity can play their part in the facial resemblance of the child to its parent. The shape of the bones constructing the air passages, especially the nasal, will in after life have the tendency to produce hypertrophic rhinitis.

DR. L. C. CLINE (Indianapolis).—I am glad to hear Dr. Shurly, because I believe him a past master in the work, and what he says is always listened to with great interest because we know he is always telling what he believes.

Dr. Shurly has certainly given us an unique paper, one that contains much original thought and I am sure we all profit by having heard it, coming from one who is so rich in experience.

DR. HAL FOSTER (Kansas City, Mo.).—I have been very much pleased with Dr. Shurly's report and feel well repaid for coming here just by hearing his paper. I agree with him in his remarks about children. I think the specific exanthematous diseases leave a great many troubles along this line. Influenza is a prominent cause of this trouble, in fact, a very common cause. Where the winds and dust blow through western Missouri and eastern Kansas we have the atmospheric conditions favorable for hypertrophic rhinitis. I have noticed that where those of the colored race are subjected to the same conditions they have this trouble just as often as the whites. I think most of the men working in our line, where colored people live in large numbers, will bear me out. As regards troubles of the throat and nose they seem to be just as susceptible as other races under the same hygienic influences.

DR. ALBERT E. BULSON, JR. (Fort Wayne, Ind.).—I have been very much interested in this paper for the reason, as already stated by a previous speaker, that Dr. Shurly is a master in this line and always presents something worth considering. In my judgment hypertrophic rhinitis is largely a result of civilization, or, in other words, the condition under which we live. It has been my experience that the cause in a large majority of cases coming to me for attention could be traced to the vitiating influences to which the patient is subjected. One of the frequent causes of the disease is over-heated houses. Our American homes are over-heated and ill ventilated to a greater extent than those of any other nationality. No one can live in a temperature of 80 degrees, as usually found in our American homes, and not suffer to a more or less extent from catarrhal inflammation of the upper respiratory tract. Not only does the increased temperature have a baneful effect upon the mucous membrane, but it reduces the resisting power to cold, and the individual is thus more subject to acute inflammation as a result of exposure. Local conditions are frequently a caustive factor in the production of the disease, and here in Indiana we find natural gas, so much used as a fuel, an irritant which aggravates, if not causes, the disease. Diet also has much to do in aggravating the condition,

those who partake of alcoholic beverages and rich foods being more susceptible to the disease than others who live plainly. In my experience individuals who live practically an outdoor life, and who belong to the poorer classes, are less subject to the disease than those who live under better conditions with warm houses and have a better quality of food. While inherited tendencies may frequently have some bearing upon the causation of the disease, yet I am inclined to think that the disease is more frequently acquired as a result of the conditions under which we live.

DR. WM. L. BALLENGER (Chicago).—I wish to ask Dr. Shurly to bring out a few points: I noticed that in his classification of the etiologic factors, he did not mention the mechanical, that is, anterior nasal obstruction. This leads to the condition known as hypertrophy. That is a factor that is surely recognized. I also wish the doctor would state what is meant by "fake foods."

DR. SHURLY (closing).—I see that I made a great mistake in shortening the paper too much; for I have not made it clear to you that I meant to have a division between predisposing and existing causes. I did not mean to say that this race factor was the only cause of hypertrophic rhinitis, but merely predisposes to this condition. I agree with the former speaker that there are very few cases of actual rhinitis and few of inflammatory hypertrophic rhinitis. It is a term used for many years for hypertrophy of the mucous membrane or bone. It is a histological process and produced by an irritation, either external or internal, and a continuous histologic process which takes place. We suffer chiefly because we have deformed nasal passages as compared with other races and animals. While the Caucasian child has a better chance for a useful life, on account of a better cerebrum, he has a less immunity on account of the inferior quality of his special senses. He has not the same quality of the sense of smell or hearing that the Malay or Indian has. The Negroes we come in contact with here, having been for a long time under the same environment, are not really typical; for it you will look at them you will see that there is a great difference in the anatomical formation of their upper respiratory passages also. The Negroes in Detroit, coming to our clinics, are very poor and have not the same capabilities or opportunities as the white man, yet I have seen few cases of hypertrophy in them, as compared with the whites. However, they have comparatively plenty of space and a better chance for the development of the mucous membranes in their nasal passages, and for breathing.

It is measles, diphtheria and such diseases which our children

acquire, and which our aboriginal ancestors did not have, that cause much of the chronic conditions. We are increasing in these causal factors, and while we are superior to them from a mental standpoint, yet physiologically speaking, we are inferior to them. We have nothing like the power in our special senses that they had. The theory suggested is that simply on account of this change from the original type, we have certainly had impressed upon us through an evolutionary process, and combined with the exciting causes that beset us, more liability to hypertrophic rhinitis. We find that the man who lives out of doors all the time scarcely ever has hypertrophic rhinitis. I did not mention the stoppage of the anterior nares because I did not want to take up the pathology of hypertrophic rhinitis.

THE PRINCIPLES OF RHINOLOGIC PRACTICE.

BY EDWIN PYNCHON, M.D., CHICAGO.

Among the textbooks which the medical student possessed a third of a century ago was William's Principles of Medicine. In latter-day medicine the principles of practice are not in evidence as an independent proposition, but are embodied in the broader field of practice itself. Notwithstanding this present-day custom, the former plan appeals to the reason as being both rational and wise. With this line of reasoning I have thought it might be instructive to give some attention to the Principles of Rhinologic Practice.

In a general way, it may be said that idealism is the goal aimed at in the practice of medicine. Surgeons, in all departments of practice, aim to get the diseased or deformed part in such condition that it will physically conform as nearly as practical to the normal. Every effort of the dentist is to get the dental arch, and each of its individual teeth, and their surroundings, to conform as nearly as possible to the ideal standard, or to the set of perfect teeth which he ever has pictured in his mind, and in order to do this he drills away decayed tissue and fills the cavity preferably with gold, so the ideal form is preserved; he straightens the teeth when irregular, and, if necessary, extracts any that are superfluous, with the object of getting the arch to conform in shape to the ideal standard; and, furthermore, when the gums are diseased he applies treatment thereto, with the same general object in view. Later on, when the dental loss is greater, he resorts to crown or bridge work, and eventually to artificial dentures, each and every step being with the same object in view, viz.: That the resulting work shall imitate as nearly as practical the perfect teeth of ideality.

The emmetropic eye is a perfect optical production, and as the eye is mated the pair should work in unison. Lack of balance or parallelism, known as insufficiency, or intrinsic optical defect, constitute the causes for the refractive errors which contribute so largely to the financial support of the ophthalmologist. In his work of correcting these errors, largely by the aid of lenses, his aim is to neutralize the optical defects and cause the eyes as nearly as possible to simulate

the emmetropic standards of perfection. Similar examples could be cited in other fields of medical practice.

In the same way, the writer is an advocate of idealism in rhinologic practice, and as structural deformity of some kind will be found present in the noses of all, or nearly all, who apply for relief from nasal troubles, the rhinologist will do his patients the greatest amount of good by taking such steps as will cause the nasal passages to resume the conformation and patency of the ideal standard. By this statement is not advised an invariable and wild rush to surgical steps, but there is contemplated the employment of all topical, systemic or hygienic means which will singly or jointly contribute to this end, remembering that improvement of the general health always affects favorably the conditions of local parts.

As a preliminary step, in order to follow the principle of practice outlined, it becomes essential for the rhinologist to decide the question as to what constitutes an ideal or perfect nose. It is quite evident, from the varying methods of treatment recommended by different authors, that there is not an unanimity of opinion as to this matter, or else its consideration is neglected. Until this moot matter is settled, so the perfect nose is an accepted fact, the same as is the perfect set of teeth with the dentist, or the emmetropic eye with the ophthalmologist, the rhinologist will be at sea, as the mariner without a compass. On the other hand, with this question settled, rhinologic practice will be elevated from the plane of empiricism to that of scientific exactness.

The writer has for a long while had his idea of the perfect nose, and has in a previous paper, written several years ago, outlined his understanding thereof,¹ hence liberal extracts will be made from this alluded to paper, as the observations of recent years have but slightly modified the original proposition. In the ideal nose the septum is practically plane, and vertically divides the organ in two passages of equal calibre, which passages have jointly a sufficient capacity to at all times easily supply the requirements of nasal respiration.

While the septal wall is plane, the outer wall in either nostril carries three turbinals or scrolls, which are practically horizontal when the patient is upright, and which, by their tortuous convolutions, add materially to the area of mucous membrane lining the passages. The openings to these passages, both anterior and posterior, being less in area than is a cross-section at any intermediate point, a slight obstruction near either end will often cause more annoyance or stoppage than will even a larger growth when located elsewhere in the

passage, and, more particularly when at the anterior end, owing to the mobility of the *alæ nasi*, by impeding inspiration will tend to induce alternate rarefaction and condensation of air in the post-nasal space, which is probably an etiologic factor in the production of posterior white hypertrophies of either the turbinals or upon the vomer, as well as a recognized cause of tubal congestion. In fact, aspiration has much to do with the causation of anterior turbinal intumescence. A feature of particular and vital importance possessed by a normal nose is that no two opposing surfaces therein ever touch, hence the ventilation in all parts thereof is always free. With these physical qualifications as outlined, the drainage will also at all times be perfect.

While the septal wall of the nasal passage in an ideal nose is practically a straight line, which it will be remembered is a characteristic of a perfect highway, built in conformity with the geometric principle that a straight line is the shortest distance between two points, still, as with the highway, which is serviceable even though it possess moderate grades and gentle curves, so long as its surface is of sufficient width to meet the demands of its traffic, so with the nose, for whenever its calibre at all points is sufficient, it will meet the demands of respiration, even though the septal line be not straight, so long as the prominences are not abrupt, nor too great, and so long as opposing surfaces do not touch, and thus impair its essential features of ventilation and drainage. There is thus additionally given proper vocal resonance, for the free and unobstructed nose may be regarded as the sounding-board of the voice.

Ample "breathway" has been regarded as the chief requirement by many rhinologic writers. It is apparent that more than this is required, for excessive space at one portion of the passage does **not** atone for a stenosis at another point. The essential requirement is that the circulating air can penetrate all portions of the nasal fossæ and at all times. The turbinals, through congestion, become more or less distended and require sufficient space in which to attain their maximum size, but should never at such time touch either each other or any opposing surface. The inferior turbinal, being the most erectile, requires the greatest amount of surrounding space. When most reduced in size, as when the patient is in a warm room, or in the examiner's office, there should be about it a space of from one-eighth to one-quarter inch, and, as the anterior end thereof is the most erectile, it is at this end where the greatest space is required. Between the middle turbinal and the septum, as seen by anterior rhinoscopy, there should be a space ranging from one-twentieth to one-

twelfth inch, for the variation at different times in the size of this body is slight as compared with the inferior turbinal.

Under the stimulus of either cold, dust or irritating vapors, the turbinals become more or less distended, so as to diminish the lumen of the nasal passages, and thus cause the inspired air to pass through in a thinner column, whereby it is the better warmed. Simultaneous with the swelling of the turbinals, the nasal secretion is increased in quantity, so as to better humidify the air, and also attract the dust or cause its precipitation. Furthermore, the evaporation of this secretion is more rapid, owing to the increased rapidity of the air current through the diminution of the calibre of the passages from the swelling previously alluded to.¹

It has been noted that the septum should be practically plane. Any material variation therefrom produces in one nostril or the other points of prominence against which the inspired air strikes with more force than when the surface is plane. As a result, the mucous membrane at such points is chronically irritated, and often dry, and invariably shows irritation by an increased redness. The further forward, or more abrupt, these prominences are, the more annoyance produced thereby. In the normal nose, the mucous membrane should at all times have a pink color, much like the roof of the mouth, and there should be no points of excessive redness, or locations which appear dry, or seem to invite the retention of visible secretion.

Septal prominences deviate the inspired air from the course it should take, and also encroach upon the space about the turbinals so as to interfere with their physiologic congestion and thus cause respiratory insufficiency. In this way a touching or even pressure may be produced when such congestion occurs. A septum abnormally thick, and yet plane, may in the same way diminish the calibre of the passages, so as to cause nasal occlusion. The congenitally narrow nose is the most unsatisfactory of all to treat.

Abnormalities of the nasal septum are, in adults, the most common causes of nasal obstruction, and their correction, when feasible, is to be always preferred, instead of making destructive attacks upon the vital turbinals. Very slight prominences, when well forward, which would seem to be absolutely harmless when turbinal congestion has subsided, and when the opening to the nostril is distended by the use of a speculum, are frequently the cause of chronic irritation, as has been often proved by the beneficial results following their destruction.² Perforations of the septum are another source of annoyance, and are generally well forward in the cartilaginous portion thereof,

and are not specific in character, but have been caused by picking the nose when a deflection exists. After the perforation is produced, its chief annoyance, when well forward, comes from marginal dryness or scabbing, and is principally due to a prominence upon or thickening of the septum close by, which, when destroyed, so there remains only a perforation in a plane septum, it will be found to give but slight annoyance.

Next in importance to septal deformity as a cause of nasal symptoms may be mentioned hypertrophy of the anterior end of the middle turbinal, so it impinges against the septum, causing either simple contact, or contact with pressure, and which alone, or in combination with septal prominences,^{3 4} occludes the attic of the nostril, and by impairing its ventilation induces catarrhal sinusitis, which through neglect may in time become empyemic, and which is the great cause of post-nasal catarrh. When pressure occurs in this region of the nose, various reflex symptoms may become manifest.⁵ The enlarged middle turbinal may also extend downward, and thus press against the inferior turbinal, so as to occlude the middle meatus, thereby affecting the sinuses connected therewith. Cobb⁶ has called attention to this fact, and that the under scroll of the middle turbinal may be so large as to not only press against the inferior turbinal, but also form a gutter, which conducts the discharges from the antrum of Highmore, the anterior ethmoid cells or the frontal sinus, so as to reach the naso-pharynx and appear as a post-nasal catarrh. In such case there is no discharge visible by anterior rhinoscopy.

It is now known that the course of the air current during respiration is largely through the upper part of the nose.^{7 8} The horizontal plane of the anterior openings to the nose, as well as the curve of the pharyngeal vault, both contribute to this end. In its passage through the upper part of the nose, owing to the greater narrowness of the passages therein, it is forced to pass in a thinner stream, and is therefore the better warmed. Furthermore, the diminution in the volume thereof tends to increase its speed, the same as the slow current of a broad stream increases in velocity when passing through a narrow gorge. A patient with attic occlusion will often complain of a sensation of inability to breathe through the nostril, even though the lower portion thereof appears free. As the openings to the accessory sinuses are all located in what may be called the attic of the nose,⁴ and as these sinuses are each ventilated through a single opening, such ventilation is best secured by the to-and-fro motion of a more rapid air current, which is happily attained in the way previously outlined.

Another disadvantage of attic occlusion is that all the inspired air is forced through the lower portion of the nose, which thus tends, through excessive stimulation, to induce congestion, and, in time, hypertrophy of the inferior turbinal, hence the importance of correcting attic stenosis. In fact, if atrophy is to be regarded as a degenerative successor to hypertrophy, then attic occlusion may be regarded as one of the etiologic features in the production of atrophic rhinitis.

During nasal respiration, as has been previously noted, the inspired air becomes warmed, humidified and freed from impurities, and the nearer perfect the nose is, the better are these functions performed. It has been assumed, owing to its increased power to become congested, that there is thrown out from the inferior turbinal the greater part of the nasal serum which renders humid the inspired air. In its passage through the middle meatus, the air current absorbs moisture from the upper surface of the inferior turbinal. It is, furthermore, quite probable that the sinuses contribute their quota of moisture, which is evaporated by the passing air current, so as to increase its humidity.

Among the defects of the nose commonly observed, occlusion may be considered of the most importance, and varies from being complete to being so slight that the patient is not conscious thereof. Frequently "the two nares are unequal in size, one being stenosed. In such case the other is compelled to do the greater part of the work, and may thus be so overworked that it cannot properly fulfill its physiologic functions. While secreting only enough nasal fluid to properly humidify one-half of the air inspired, it is giving entrance to much more than half of the air required; hence this air is not sufficiently humidified and, as it enters in too large a column, it is likewise not so well warmed. Furthermore, the volume of air entering the more roomy nostril tends to dry the mucous membrane therein to an abnormal degree and is harmful; therefore, it is as essential that the normal nostril shall not be too roomy as that it shall have adequate patency.¹ A stenosis of one nostril will frequently cause hyperemia of the other nostril, even though it be free from structural defect, and contact in either nostril generally means contact at some point in the other nostril, hence alternating stenosis is frequently noted," as well as a periodic susceptibility to attacks of acute coryza.

One of the most important of the nasal defects is the condition wherein a partial stoppage of either one or both nostrils exists, though the same is *unrecognized by the patient*, and any or all reflex symptoms or troubles present, and due thereto, are attributed to other

causes. In this condition, there is a touching of opposing surfaces at one or more points, and at the same time a compensatory increase of size elsewhere. In such case, upon inquiry, it is learned that certain reflex or secondary symptoms are complained of, which the experienced rhinologist will at once, and correctly, attribute to the defective nose.

Next in importance to occlusion, and largely secondary thereto, owing to impaired ventilation and drainage, we have excessive or abnormal nasal secretion. Nasal obstruction, particularly when contact of opposing surfaces exists, interferes with the normal evaporation of the nasal muco-serum, which is normally secreted by the nose to the amount, approximately, of one pint a day. When evaporation is interfered with, or prevented, the retained secretion becomes more or less inspissated, and, through retention, is irritating to the mucous membrane at that point, causing further secretion therefrom to be abnormal and thickened, so it cannot be easily evaporated, as is the case with the normal nasal serum. As a result of long-continued irritation, tissues in the nose, particularly of the turbinals, may become chronically relaxed and baggy, so as not to contract under cocaine, being a true hypertrophy or degeneration, and, as such, give out increased and abnormal secretion, and must, therefore, be surgically attacked. The systemic effects upon both the pulmonary and gastro-intestinal tracts from the descent of catarrhal secretions are only too well known.⁹

As the tenor of this paper is to emphasize the fact that structural deformity is almost invariably present in diseased conditions of the upper air passages, it is an easy step to infer that the chief indication is to cause the several parts to assume as nearly as practical the contour and character of the ideal standard, which implies the removal of all obstructive, redundant or pathologic tissues, the correction of deformities, and, in the nose, to secure appropriate space between all opposing surfaces. Of course, each case is a rule unto itself, and hence practicability must be the guide. When this much is done, it will be found that Nature will do the rest, in a great majority of all cases presenting themselves for treatment.

Preceding the surgical work, certain preliminary steps should be taken. The specialist should not blind himself to mal-conditions outside of his particular field, hence, if there be a call for systemic medication, it should be administered. Attention should also be given to the patient's hygienic surroundings and habits of life. As cleanliness is the keynote of modern surgical practice, the nasal toilette should be well looked after, which, in the writer's experience,

is best accomplished by the hourly sniffing of a bland alkaline solution of the same specific gravity as the nasal serum, which is 1015°, and according to an exact method elsewhere described,^{10 11} which can be followed, unless marked occlusion exists. This alkaline solution is regarded simply as a cleansing agent—a suitable soap for the mucous membrane of the nasal passages—and contributes only in this way toward a cure, and its hourly employment is in line with the known efficacy of the small dose, frequently repeated. In case of atrophic rhinitis, the sniffing medicine will have to be supplemented for a while with the use of a tepid nasal douche, twice or thrice daily.

The occasional use of a suitable pocket inhaler¹² will also assist, as vapors more readily penetrate the higher and narrower recesses. The systematic use of this home treatment advised, in addition to a few office treatments with sprays and pigments, will in a week or ten days' time prepare the nose for operative steps, and will often cause the subsidence of points of intumescence, which at the time of the first examination seemed to require operative attacks. The use of the alkaline sniffing medicine is to be kept up during the entire course of treatment, and is as efficient in keeping the parts clean after operations as it is in the preliminary preparation.

A word must also be given to the question of blowing the nose, which, when correctly done, is one of the most efficient methods of cleansing. Use of the handkerchief is undeniably more esthetic, and will do as a makeshift at times, but, for thorough cleansing, the nose should be blown without the use of a handkerchief, and at first with both nostrils open, a short vigorous expulsive blow being given, and, if necessary, repeated two or three times, the patient meantime stooping forward over a wash-basin or sink, with head thrown well back. After this the head should be inclined forward and each nostril blown alternately by closing the opposite one, in the usual manner, only much less force must be employed than when both nostrils were open. In this way there is no danger of injuring the ears if the nostrils are not too much occluded, and the great advantage lies in the fact that by this method air is made to pass so quickly by the openings of the sinuses that if there be catarrhal secretion therein, which is so often the case, it will thus be drawn out and expelled. This method of blowing the nose may be practiced two or three times a day, and will generally insure a free nose for several hours, or one which can be easily cared for by the handkerchief.

During office treatments, compressed air, either plain or in the

form of a nebula, properly directed through a long, slender and slightly curved tip, particularly after a partial removal of the middle turbinal, will often prove more efficient in cleansing an empyemic sinus than will an aqueous injection. Recent experience with heated air has given promise of its great value in all cases wherein either hyperemia or excessive secretion is observed. An advantage in the practice of Politzerization, derived from the air douche, is its cleansing effect upon the nasal passages, and when, at the same time, both external auditory canals are tightly closed, a nebula may be easily driven into the sinuses, thereby giving magical relief from the oppressive headache, due to sinus occlusion, which often accompanies acute coryza.

After intranasal operations wherein the deeper structures are wounded, I have found, in addition to the home treatment, that a one per cent. hot carbolized douche, used from three to eight times daily for a few days, will allay the inflammation, and reduce the reaction to a minimum.¹³ Latterly, I have had made a long oval tip, which can be introduced far back in the nostril, whereby the efficiency of the douche is increased. This douche can even be employed after the slighter operations, and in order to insure its correct use, I give the patient a printed sheet of directions.

At all times, in intra-nasal surgery, it is the wisest plan to do but little at a time, and allow each wound to be well on the road toward recovery before the next operation is made, particularly when in close proximity to its predecessor. Aside from the demands of urgency in special cases, it is generally wisest to first operate those defects which are most accessible, as anterior septal deformities and tonsillar or post-nasal abnormalities, after which the tolerance of the patient is increased, so suitable attacks can be made, when required, upon occlusions farther in and higher up, in order to insure the necessary freedom in attic ventilation and drainage. In correcting nasal defects, it is generally best to select the better nostril for first attention, and after it is in satisfactory condition it will serve for respiratory purposes, while the other nostril is being operated. It is, though, necessary that all obstructions be removed, for one stone left remaining in a gutter will obstruct it nearly as much as when there were several; or, again, as a further comparison, if a leaky roof be repaired in all but one spot, the leak at this point may cause nearly as much annoyance as did the several leaks before.

In the way of surgical work, the obliteration of slight prominences of the nasal septum is often as essential as is the correction of the grosser defects.² The explanation is that, in case of the latter, com-

pensatory defects are in time created, while the slighter deformities only cause chronic irritation, which may be of many years' duration. When operating a large ridge, there is often less annoyance in removing one-half thereof at the first sitting, and the remainder two or three weeks thereafter. Thus, in some cases, after removing the lower portion of a ridge, when the mucous membrane of the remaining portion is uninjured, it holds away the opposing turbinal and helps toward quick healing. It frequently occurs that the upper margin of a ridge is soft or compressible. If so, such portion had better be first destroyed with the electro-cautery, when, after a week's delay, the remainder thereof, being bony or cartilaginous, may be removed with the saw. In fact, a slight incision with the cautery point may be made both below and above, just before using the saw, and will materially assist toward the diminution of hemorrhage.

Another time, when the operation can wisely be divided in two steps, is when removing the excessive tissue upon the convex side of a deflected septum, the concavity of which is gently curved. During the first operation the saw is held parallel with the anterior half of the concavity in the opposite nostril, while during the second operation the opening of the nose is so pulled away from the septum that the saw can be held parallel with the posterior portion of the concavity. In order to know more exactly the thickness of the parts being operated, a septometer should be used.¹⁴ By operating as suggested a simple deflection will remain which may afterward be corrected by an Asch or Gleason operation, though, when the concavity is not too pronounced it will often be found, by removing the thickened convexity, as described, that the lumen of the nostril on that side is increased to such a degree as to give comfort and permit of satisfactory respiration.

Secondary hemorrhage is liable to occur after the more extensive intra-nasal wounds, the greatest danger being within the first twenty-four hours. By the introduction of a Simpson-Bernays tampon, so it will cover the traumatic area, such complication may be averted. Before its introduction, the forward end and sides of the tampon should be smeared with vaseline, and after it is properly located by aid of sight, it may be quickly made to swell by the use of an aqueous spray. It should be removed the following day, and, if desired, can be gradually removed in sections, as its several layers can be withdrawn one after the other. Personally, I use the tampon only in special cases, wherein hemorrhage is particularly feared, and prefer generally to depend upon the hot carbolized douche, which tends to prevent bleeding.

After intra-nasal wounds involving the harder structures, as of the septum, smooth healing is materially advanced by the practice of daily massage of the wound until healed, at first gently, though later, as the soreness subsides, more force should be employed. Massage removes exuberant granulations and debris, and causes the wound to heal from the periphery toward the center, thus gradually reducing in size until healed.² Without the massage there may be reformation in soft tissue of a growth similar in form and size to the one removed. The omission of such after treatment explains the recurrence, after removal of septal ridges, as have been reported.¹⁵ My attention was first called to the value of massage by Rice,¹⁶ who advocated its use in the treatment of chronic septal ulcers, particularly in connection with atrophic rhinitis. As chronic ulcers of the septum can be best healed in this way, it seemed rational to employ massage in the treatment of intra-nasal surgical wounds.

The immediate result at the time of the operation will generally indicate what permanent result may be expected, though for several days thereafter, owing to marginal swelling, the appearance of the wound is unpromising. Occasionally, after a septal operation, there occurs a sufficient swelling of the base at the operated point, so a slight secondary operation is called for a few days thereafter, whereby such base may be more thoroughly removed. In the process of healing under massage, there are destroyed slight points of elevation, and at the same time by stimulation there is a filling up of slight depressions. Lastly, an eventual absorption of hyperplasia in adjacent prominences follows, though the maximum result may be delayed for a month or six weeks, after the healing of the wound. By this method, the resulting mucous membrane is smooth and firm, and appears in all ways like the normal.

The electro-cautery has been extensively used in the past in the treatment of nasal occlusion, and principally by cauterization of the anterior end of the inferior turbinal. While an improvement was for a time thus secured, it was soon followed by a recurrence of the old trouble. The explanation is that the turbinal congestion or intumescence was only a symptom, and the result of irritation from some other cause, generally some deformity of the septum, and often of apparently slight importance, though still sufficient to cause and keep up irritation of the opposing turbinal. When, in place of intumescence, there is genuine hypertrophy of the soft tissues of the turbinal, so it will not contract under cocaine, then a thorough cauterization is indicated, so as to remove excessive and obstructive tissue and destroy hypertrophied glands from which the secretion is excessive and abnormal.

Should the bony framework of the turbinal be involved, then a sufficient portion thereof should be surgically removed, in order to give free breathway and drainage.^{4 6 17 18} The reason why either the ridge upon the septum or the turbinal hypertrophy should be operated is chiefly the same; viz.: To improve the ventilation and drainage of the nose, and to do away with the intermitting touching of opposing surfaces, and a nice discrimination as to where the attack should be made, when both turbinal and septum are at fault, comes from increased experience. At times, turbinal intumescence, with tendency to hypertrophy, can best be overcome by the use for a few days of a nasal bougie.¹³

In conclusion, it may be added that the ultimate results following a course of intra-nasal surgical treatment are far better than are the immediate results. It must be remembered that for a time the new tissue formed as the result of an operation is delicate, and may, therefore, be compared with an infant's skin, but which, as time passes by, will become toughened and inured to climatic changes, so as not to suffer thereby, and thus a progressive improvement may be expected for a year, or even longer, after the treatment has been discontinued, though such improvement may be intermitting instead of constant, being influenced by climatic conditions and the exposures which the patient may from time to time sustain.

The improvement at first obtained is sometimes not fully appreciated by the patient, as it is only compared with the condition existing before the treatment was begun, though it may be said that at that time the patient was at a Y in the road, and drifting away from the proper course, while through the treatment the direction has been changed to the right course, so in future years the proper comparison to make will be the conditions as they are, not only with what they were, but with what they might have become.

CONCLUSIONS.

1. In the normal nose the nostrils should be of equal calibre and should jointly have a sufficient capacity to at all times supply the requirements of easy nasal respiration.

2. In the ideal nose the walls of the septum are practically plane, and are at no time or place touched by the tissues of the outer wall, in either passage, and, furthermore, no points of contact exist elsewhere therein, so as to interfere with either ventilation or drainage, or prevent the normal evaporation of nasal moisture.

3. While in an ideal nose the septum is vertical and nearly plane, a moderate irregularity thereof will not impair the nasal respiratory

functions, providing there are no points of contact or abrupt elevations therein, and the lumen at all points is sufficient.

4. Abnormal redness of the nasal mucous membrane is an unfailing sign of irritation, the cause of which is generally of a structural nature, and, therefore, amenable to surgical treatment.

5. The indications for operative interference depend upon both the subjective and objective symptoms. A noticeable inadequacy of either nasal passage, the presence of excessive or retained secretions, or an abnormal redness of the mucous membrane at any point, are all evidences of abnormality, which, if coupled with inconvenience to the patient, invite corrective attention.

6. In the treatment of chronic hypertrophic nasal troubles, the indication is to remove all obstructive, redundant or pathologic tissues, and at all times the chief indication is to cause the defective nose to conform as nearly as practical to the contour and character of the ideal standard.

Columbus Memorial Building.

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DISCUSSION.

DR. BARNHILL (Indianapolis, Ind.).—Most of the principles laid down by the doctor I would be quite willing to accept. I rise to ask a question as to what method Dr. Pynchon has used in determining the distance of the middle turbinate from the septum. I do not believe that I have personally seen a case in which I could determine this accurately and it has seemed to me that there must be at times a normal condition in which the septum does lie in slight contact with the middle turbinate. In the New York Rhinological Society there was a discussion as to this, and at that time some of the best men of this society believed that there was some normal contact between these two parts.

DR. PYNCHON (closing discussion).—In reply to Dr. Barnhill, I would say that of course it is very difficult to decide accurately about this matter of the middle turbinal, but I have frequently noticed in patients who come to me that there is a lack of proper space, and when I have secured this by surgical means so I can pass a small cotton pledget through with ease from front to rear, it allays the symptoms complained of to a marked degree.

While I have talked about the perfect set of teeth and the emmetropic eye, I suppose they are not often found. It is the same with the nose. I cannot call to mind that I ever ran across a pair of perfect nostrils, but I have occasionally found one perfect nostril wherein I could tell by sight or by the use of the cotton applicator that there was no contact at any place. In hay fever there is a great weakness for this middle turbinal being too close to the septum.

SUPERHEATED MEDICATED AIR IN DISEASES OF THE EAR AND NOSE.

BY JOSEPH C. BECK, M.D., CHICAGO.

In making my report of over one year's experience with the superheated medicated air in the treatment of certain affections of the ear and nose, I desire to state at the outset that I do not claim originality of its use, nor do I claim that it is a panacea for all ailments and not the only treatment in the ear and nose affections. In fact, I have been unable to cure all my cases with its use, and bring an adverse report of its use in conditions in which it has been advocated, and great curative effects claimed, namely, in chronic catarrhal conditions of the middle ear.

I believe, however, that I have improved the method by which hot air can be applied and combined with medicinal substances, so that the results will prove to be superior from this agent than formerly; at any rate, I have found it a great adjuvant to the other already accepted methods of treatment.

Heat has been used in ear diseases as a therapeutic agent mainly to relieve pain, to stimulate absorption of pathological products, like connective tissue formation, or other deposits. It is used very frequently to promote suppuration in other parts of the body. I have employed it with this purpose in chronic suppurative processes in the ear.

The air itself serves as a drying agent. It also serves as a conductor for the medicinal substances.

Medicinal substances in gaseous forms and vaporized have been used in the diseases of the ear, as, for instance, chloroform, turpentine, and iodine.

The combination of these three agents, namely, air, heat and medicaments, I have applied in my experiments and treatment of my cases.

In order to make the heating and application of the air easy, I have devised a little air heater, which consists of a metal cylinder mounted on a handle. The tip of this metal cylinder is mounted by a wood mounted by wood fiber canula, two inches in length, which is de-fiber canula, two inches in length, which is detachable. Within

this cylinder is an incandescent lamp, which carries a current of 115 volts, supplied with a switch at the handle to turn off or on the current by simple compression. At the back of this cylinder is an opening into which is inserted the nozzle of the compressed air tube. The medicator is within the cylinder; it is a small metal box, containing a piece of felt, which is saturated with the desired medication, and it acts at the same time as a filter for the air.

As soon as the electric current is turned on, the metal cylinder begins to get hot, and within a minute or two it has acquired a sufficient heat to warm the air which passes through it from the air tube. As soon as the current of air is allowed to pass through this heated chamber, it will come out in a concentrated warm stream from the tip of the wood fiber canula. It requires about two minutes to produce a very hot current, but the temperature can be regulated by the switch. The pressure of the air can also be regulated by the cut-off on the air tubes.

After experimenting with about fifteen different volatile medicinal substances, I have selected three which give the best results, namely, formalin, menthol and chloroform.

EXPERIMENTS.

Thermal. 1. By making contact for a half a minute, the temperature of the air current is brought up to the desired warmth, that is, 120 degrees F.

2. By keeping on the contact for six minutes, allowing the air to pass continuously, we get the maximum temperature, that is, 175 degrees F.

3. Making contact without allowing any air to pass through the heater for four minutes, we obtain then an air current of 265 degrees F.

4. It requires four minutes for the heater to cool by allowing the cold air to pass through when contact is taken off.

5. By making contact every half a minute and allowing the air to pass continually, we obtain a constant temperature of the air of from 120 to 130 degrees F.

BACTERIOLOGICAL EXPERIMENTS.

I will simply read the titles of the experiments; the detailed results will be published later. Suffice it to say, however, that the heated formalin filtered through a piece of felt in the manner described in the paper has distinct bactericidal properties, as these cultures show.

1. Cultures made of ordinary cold unfiltered compressor air.
2. Of filtered cold compressed air.
3. Of hot unfiltered compressed air.
4. Of hot filtered compressed air.
5. Of cold unfiltered formalin compressed air of 5, 10, 20, and 40 per cent.
6. The same, filtered.
7. Heated formalin compressed air unfiltered, at the above strengths.
8. The same, filtered.
9. Passing cold formalin compressed air at the above strengths over pure cultures of (a) staphylococcus; (b) streptococcus; (c) tubercle bacilli; (d) anthrax, and cultures made of them after that.
10. Same experiments with heated formalin.

For the application of this method of treatment, I have selected a number of affections of the ear and nose, but principally cases of chronic suppuration of the middle ear, and I shall describe the latter first. I divide the cases in two groups: (a) Those with bone involvement. (b) Those without bone involvement.

The differential diagnosis of these two conditions was made by the method of segregation, that is, washing out the cavity of the middle ear with sterile water and boric acid, by means of a Dickerman's canula. The washings were centrifuged and the sediment examined microscopically for bone dust and cholesterin, and cholesteotoma cells. The findings from these examinations were of great value for our prognosis. Where bone dust or cholesteotoma cells were found, the prospects for a cure were not encouraging. If, however, the findings were negative, the outlook for a cure was very promising, and even if bone dust was present, I am glad to state the results were far superior to the ordinary methods.

The application of this method of heated medicated air has a three-fold use.

1. It stimulates suppuration and helps to throw off pathological processes, thereby producing a healthy surface of healing. It produces epidermization more rapidly.
2. The cavity is dried, and produces a poor culture nidus for bacterial development.
3. The formalin in this gaseous state is forced into all crevices, and exerts its germicidal action more efficiently.

In order to describe the technique, I will report two cases, as illustrations.

Case 1. Miss C., 17 years old, suffered from a malodorous discharge from her right ear constantly for more than one year. Status: Right ear, membrana tympana fairly normal in appearance; membrana Schrapnelli has in its center a small perforation, through which oozes a small quantity of malodorous pus. The head of the hammer is visible, and a fine probe gives evidence to bare bone. A smear preparation shows but a few micro-organisms. Examination of sediment washings demonstrates bone dust. There are no marked obstructive changes in the nose, and the Eustachian tube is clear. Hearing is only slightly abnormal.

The usual method of treatment was carried out by myself for four months, without any improvement in the condition. July, 1902, after the usual cleansing and drying, I applied for the first time hot air with formalin, as follows: I directed the canula of the heater to the opening of the Schrapnell membrane, and allowed to pass into it a current of air of ten pounds pressure, at a temperature of 120 degrees F. The felt was saturated with a 40 per cent solution of formalin. The air was allowed to pass in it for three minutes, then the canal was gently packed with a gauze strip, and the patient sent home. The next day the ear was examined, she did not complain of pain, but the condition was not markedly changed. The treatment was repeated for four more days; the patient complained of some pain, and the discharge was more profuse, so that she took out the gauze packing, which she claimed was blood-stained. I found evidences of blood in the canal, but there was less odor to the discharge. From this day on, improvement followed, so that after three week's daily treatment, the discharge entirely disappeared. Anticipating, however, a return in these very unfavorable cases of perforation into Schrapnell's membrane, and the rarity of a cure without at least removal of the ossicles, I ordered the patient to return every month for examination, which she did, and I am glad to say that up to date there is no return of the discharge.

Case 2. Miss L., 26 years old; had running ear since childhood. The discharge would stop at times for a month or two, but always returned. Status: Pus abundant, and of a bad odor.

Microscopic examination.

Washing and sedimentation show no bone dust or cholesteatoma. I had been unsuccessful in the improvement of this condition after two months' treatment of the usual method. I, therefore, applied the super-heated formalin air daily for five weeks, in precisely the same manner as in case 1, except that the time of application varied from five to ten minutes. The usual treatment of nose and throat

was carried out at the same time. The discharge entirely disappeared after five weeks. Five months have now elapsed, and no recurrence has yet taken place.

I have applied the superheated medicated air in twenty-three cases of otitis media, *catarrhalis chronica*, as described by Hopkins and Oaks, for the relief of deafness, and tinnitus, and I am sorry to state that I have not obtained satisfactory results; in fact, three patients have complained that whenever heat was used, the tinnitus was aggravated.

The use of heated air passed into the middle ear by means of the Eustachian cavity has given me the following experimental result:

A case of atrophic rhinitis, with a large perforation of the tympanic membrane was the subject. A thermometer was passed as far as possible into the meatus externus, and then the opening was plugged around the thermometer by a compound used by dentists, then a wood fibre catheter was warmed and passed into the Eustachian tube. The hot air, at the temperature of 120 degrees, was then allowed to pass into the catheter for more than seven minutes, until the patient complained of a hot sensation about the Eustachian opening. The thermometer did not show the slightest rise in temperature, and from this I concluded that the hot air introduced into the middle ear by this route is of no value, having cooled off before it reached the middle ear.

Other conditions for which I have used hot air by this method were acute salpingitis, earache in children, acute otitis media (in some, perforation followed), furunculosis, weeping eczema. In this latter condition, the results were gratifying. As a rule, itching was relieved, and the weeping eczema rapidly cured.

The superheated air in the treatment of nasal affections I have applied in two conditions: (1) Acute sinusitis without pus; (2) lupus of the alæ of the nose.

In the first stages of acute sinusitis with much pain, I have employed the heated air, medicated with menthol, of full strength, at the temperature of 105 degrees F., for ten minutes, the canula having been replaced by an olive tip. Treatment gave most satisfactory results. In one case of a physician whom I treated for three days by the usual method of mentholated steam inhalation and other local and general treatment was not relieved, but after one application of dry heat for ten minutes, the condition was very markedly improved. This may have been accidental, but in conjunction with my experience in other cases I am confident that dry hot air is preferable, and was the cause of the rapid change for the better.

In the case of lupus of the ala, I applied the formalin heated air, of a temperature of 230 degrees F., ten minutes at a time, for ten sittings, when the condition was perfectly healed. Lichtwitz reports a similar case, with same result, with the use of Hollander's apparatus.

In conclusion, I desire to say that not all cases which I treated with this method got well, as my full report of over thirty cases will show, nor that the cases of chronic suppurative ear disease which are now dry are permanently cured, but I have found this method an excellent addition to the non-surgical treatment of chronic suppuration of the middle ear, the condition which makes up such a large percentage of our cases.

Even when clear indications for operative interference exist, the patient will not always consent to it, and I believe that in such instances this method may be given a trial.

REPORT OF CASES.

Fourteen cases of otitis media catarrhalis chronica, varying from the age of twenty to fifty-four years, all having been complaining between one and ten years of deafness and noises in their ears. All gave a history of naso-pharyngeal trouble. All have a Rinné negative, and whisper reduced in varying degree. Improved some in hearing right after inflation, but this is not lasting. All were treated by the usual methods for from six weeks to six months, without any improvement, then added the use of superheated air to the tolerance of the patient, which was about 150 to 200 degrees F., for three to five minutes. Treatment lasted from six months to a year, twice a week. The general complaint during and a short time after the treatment was dizziness, in all the cases. There is no improvement in the hearing of any of the cases, the tinnitus was relieved in most of the cases, and three of the patients give a history of increase in the tinnitus after the treatments.

Fourteen cases of otitis media suppurative chronica, ranging from the age of nine to forty-three. All have had running ears for more than a year, and most of them for several years. The washings from the middle ear were sedimented and examined microscopically, and in five of the cases showed absence of either bone dust or cholesteotoma cells, while nine showed bone dust, but only three of those showed distinct epithelial cells of cholesteotomatous nature, one giving the distinct chemical reaction. All these cases were treated previously by the usual accepted methods, with varying success, then using additionally superheated formalin air, with marked improvement.

Seven cases were cured, and remained so, the longest seven months, the shortest three and a half months. Of the remaining seven, five were markedly improved, and are still under treatment. Two were operated on (radical mastoid), and one ossiculectomy. Both still discharging and showing bone dust and epithelial cells microscopically in the sediment washing. All these cases are in private practice. Many cases treated clinically, but no record could be kept, owing to the irregularity of the coming, and many would stay away after a short period of treatment.

ACUTE OTITIS MEDIA (EAR-ACHE).

Four cases, all in children from three to seven years of age. Distinct history of adenoids. All relieved of pain after one or two applications of heat for three minutes, at the temperature of 120 degrees F. Three of these cases perforated, getting well by the usual mode of treatment after two to four weeks.

OTITIS EXTERNA FURUNCULOSA.

Two cases. Relieved of pain after the application of superheated air at the temperature of 200 degrees for five minutes, but incision was necessary to obtain a cure.

ACUTE SINUSITIS WITHOUT EVIDENCE OF PUS.

Three cases, in adults, using menthol, formalin alternately, in five per cent solution, the latter being very irritating. Every day for five minutes, for three days. All three cases very much relieved after treatment.

LUPUS OF THE RIGHT ALA.

One case; man, thirty years old; distinct lupoid tubercles treated for a period of six weeks by X-rays, with not marked improvement, and after eleven applications of superheated air, at a temperature of 255 degrees, the condition commenced to show improvement, and healing followed.

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DISCUSSION.

DR. J. J. KYLE.—I want to express myself in favor of using super-heated air in the treatment of catarrhal conditions of the middle ear. I have been using it for the past year exclusively. I believe where inflation of the middle ear is indicated the use of cold air predisposes to congestion. The super-heated air will bring about a hyperæmic condition rather than congestion. This hyperæmia, if continued day after day, stimulates the absorption of the exudate. We thus bring about a very good result, especially in old catarrhal conditions, where as by the injection of medicated solutions such as pilocarpin, we are in danger of producing a good deal of inflammation. With the use of super-heated air, we are better able to control the amount of hyperæmia, judging the condition by the ocular appearance of the membrana tympani. So far as the relief of tinnitus is concerned, I have observed no flattering results from the use of super-heated air.

DR. J. A. STUCKY (Lexington, Ky.).—Perhaps no subject comes before us in which we are more interested than the treatment of the chronic suppurative conditions of the middle ear. If we can get sufficient heat there it is undoubtedly the ideal treatment. I would like the doctor to tell us how he knows how much heat he has. Does he depend on the sensation of the patient?

DR. PYNCHON.—I have used an air heater quite a good deal and one of its defects which I have noticed is what Dr. Stucky refers to. It has occurred to me that if there could be put around this instrument one of those little circular thermometers it would be a good thing. They are not absolutely accurate, but one could easily learn what temperature it would register and it would thus be a guide which would be in front of the eye. This could be easily applied to the instrument.

It seems to me one of the principal indications for the use of hot air is pain. It is of more value in pain than anywhere else.

DR. VAIL.—I have not used this method of treatment to any great extent. I used the electric device made by Meyrowitz of New York years ago, but I could not see that the current of hot air directed on the drum head had any effect. I have found the inflation of warm air from Politzer's blow bag to be a very useful and grateful method of treating acute catarrhal cases of otitis media. I can appreciate from my own standpoint the difference between hot and cold air in the middle ear. I once had an attack of acute catarrhal otitis media and was Politzerized with warm air. The treatment was very grate-

ful. I felt better immediately. I sometimes keep my blowbag on the radiator where it is always hot, instead of in a drawer, where it more or less cold. I still use the old Politzer method of inflation in these cases; it is safe and gives the best relief. I use the catheter in chronic cases, but prefer the Politzer method in acute troubles.

DR. BECK (closing discussion).—The difficulty in the Eustachian tube route is that the catheter is too hot and the patient cannot stand it, and the wood fibre catheter was selected in order to control the heat. In a case of atropnic rhinitis I found the heat was borne much better than ordinarily. In reply to Dr. Stucky, in the experiments I made, will say I took a chemical thermometer, made an artificial external canal which I occluded like the middle ear. Then the instrument with the tip was passed into this canal. (Reads from paper.) "In a case of atrophic rhinitis, with a large perforation in the tympanic membrane, etc." I take the tube and put a thermometer at the point where the heat would strike the ear. I was in hopes Dr. Vail would discuss the paper at length, for I selected him as the victim to try my instrument first.

NASO-PHARYNGEAL FIBROMA—EXHIBITION OF SPECIMENS AND NEW FORCEPS.

BY J. A. STUCKY, M.D., LEXINGTON, KY.

Fibromata of the naso-pharyngeal space are entirely different histologically from edematous or polypoid growths, and vastly more serious, being extremely hard and vascular, with large base—not pedunculated, but broad and irregular—springing from the periosteum or fibrous layer. They grow rather rapidly, and, having filled the space in the naso-pharyngeal vault from which they have their origin, they invade other cavities, after causing absorption or resorption of the bony structure intervening, sending out arms in various directions along the lines of least resistance. Prof. Moritz Schmidt has found only one case in every 2438 of naso-pharyngeal growths. They are even less frequently found in this country. Of thirteen cases, seven were in males. The surface of the growth frequently ulcerates, which leads to adhesions, which complicate operation for removal. The blood supply is large, being made up of small arteries. As a result of this free arterial blood supply, the hemorrhage is violent and alarming, quickly rendering conditions for a time critical, unless very promptly controlled.

The ages of patients I have seen have been fourteen and sixteen years. Zarincho, quoted by Bensch, gives as one of the theories why these growths are found in the young, and not after the twenty-fifth year: "During the period of development, for unknown reasons, the periosteum may become unable to form bone over a circumscribed area, and the physiological increase of nutrition incident to the development of the body at this period, causes an equal amount of nourishment to be carried to this circumscribed area, which not being used up in the formation of bone, leads to hypertrophy of the periosteum at this point, and consequent development of the fibroma." This seems to be a rational explanation of the cause—and the arrest of the development of the skull after the twentieth or twenty-fifth year, when this increased nutrition ceases, explains the cessation of development of growth at this time. Bosworth reports cases of spontaneous disappearance.

The fibromata included in the report are those that spring from the vault of the pharynx and are most dangerous because of their great vascularity—the origin of this being a central artery coming from deeper structures. Karl Hirschberg regards these as “Reste,” or remains of the cord, and has reported two cases, one growing out from the fossa sphenopalatine, developing outwards and in-

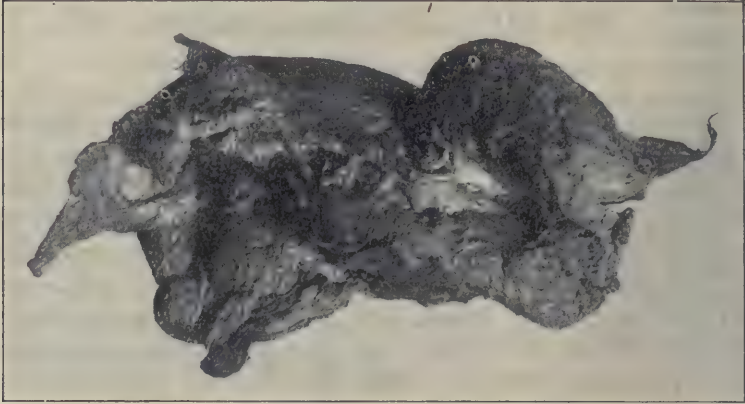


Fig. a. Showing posterior surface of attachment.

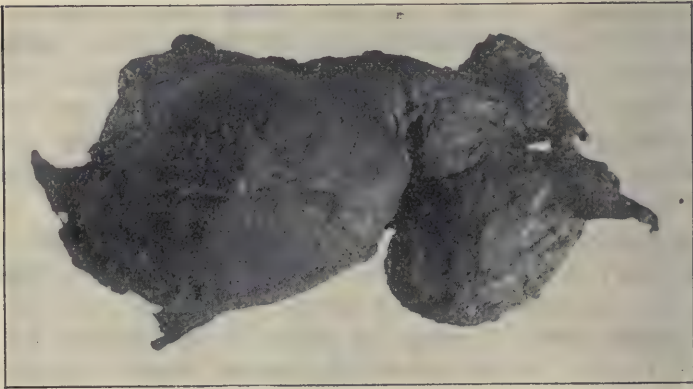


Fig. b. Showing anterior surface.

Plate I. Case I.

volving nearly one-half of the skull. The other sprang from the sphenoid bone. He describes them microscopically as being remnants of the cord, in one case having more of the appearance of sarcoma, while in the other that of fibro-sarcoma, or fibroma. He regards them as non-malignant, as they do not infiltrate other tissues. Bensch says they are “clinically and not anatomically malignant.” This I believe to be true.

Cases I have had with similar history: Mouth breather, gradually becoming worse until growth becomes so large as to interfere dangerously with respiration, but seriously impairs deglutition. Emaciation from want of nutrition, and weakness from frequent hemorrhage, made the cases, when I first saw them, most pitiable objects to look upon. In the two cases the growth protruded into the mouth, was smooth, pinkish color, lobulated, etc., was attached to the entire vault of the pharynx, one arm extending into the right nares until it protruded through the nostril, antrum wall was absorbed and antrum filled with growth. Electrolysis had been repeatedly and thoroughly used for months before radical operation was performed.

Before resorting to this, it is well to bear in mind the remark of Driffenbach in his recent text: "Great courage is required to undertake this operation, because the surgeon is almost compelled to choose between one of three conditions: Choking to death of the patient during the operation, bleeding to death at the time of the operation, or not finishing the operation." These conditions result from difficulty in reaching the seat of growth. The cold snare and cautery loop were faithfully tried, but on account of size, shape and location of attachment, were only partially successful, and in my first case, the aid of the general surgeon was called, who did the external operation. I now believe that the entire operation should be done by the rhinologist, because of the greater dexterity which he acquires by daily practice within naso-pharynx and nares. Furthermore, I believe any growth having its attachment within the nares or naso-pharynx can be radically removed through natural openings, without any external incision, by use of snare and forceps.

The first specimen I show you is from Case 1, operated upon May, 1894—R. H. aet. 15—mouth breather—increased difficulty began one year ago—rapidly grew worse—at this time nasal respiration impossible—pain in right ear—speech greatly impaired, also sense of taste and smell—deglutition markedly interfered with—right nasal cavity completely blocked up, and growth protruding through the anterior nares. Upon opening the mouth a large pinkish gray tumor completely filling the pharyngeal vault, pushing the velum palati forward until stretched to its utmost, and extending downward until half of pharynx was filled. Electrolysis had been faithfully used without appreciable result. Attempts were made to remove growth by ecraseures and snares (made for the purpose) both through the nose and mouth, but these were also ineffectual. It was impossible to put a loop around the base, and the external operation of slitting open the cheek was successfully

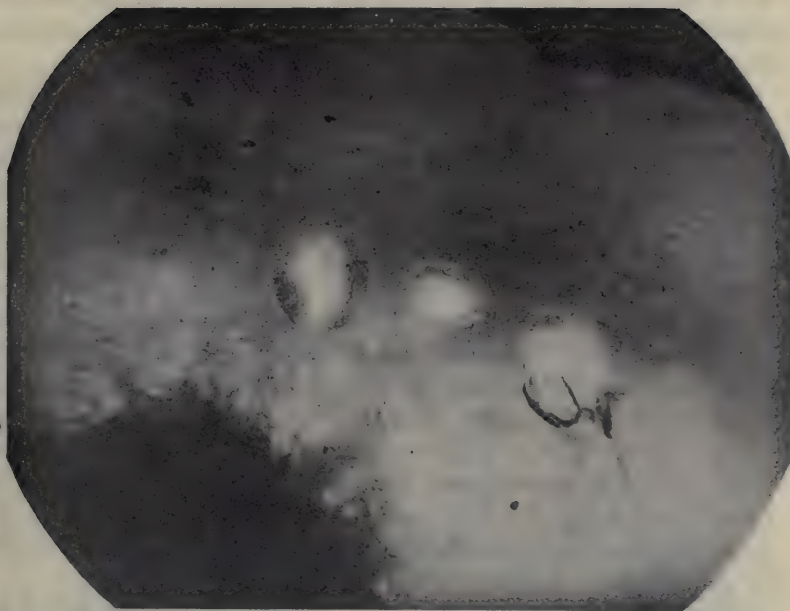
performed, though the recovery of the patient was little short of miraculous. The growth shows the imprint of wire used in snare of extra strength and size, made especially for the case. The wire was gradually tightened every two hours. This was kept



Plate II. Case II. (Exact size.)

The largest and smallest masses removed with forceps.

up for three days and nights, when the No. 3 piano wire broke without cutting through. I afterwards succeeded in removing about one-third of it through the nose by twisting and pulling with large hemostatic forceps. The external operation was after-



Plates III and IV.

Microscopic sections of growths removed from cases I and II.

wards performed by Drs. Barrow and Kinniard. The patient made a perfect recovery and a month ago showed no evidence whatever of any recurrence.

The second specimen is from Case No. 2, W. C. aet. 15. About nine months ago first noticed very great difficulty in breathing through the nose—his parents often feared he would choke to death in his sleep. Previous history of nasal discharge; Frequent attacks of sore throat and recent attack of measles and whooping cough; since then the tumor, which now filled the entire vault of pharynx and left nostril, had grown very rapidly. Examination showed same conditions as in Case 1, except the left nostril was involved and not the right. In both cases, adhesions were very abundant, to the septum, turbinates, velum palati and posterior wall of the pharynx. In this case no attempt was made to use the snare. A pair of cervix forceps, borrowed from the gynecologist, were used. The growth was firmly grasped through the mouth, as close as possible to its attachment to the vault of the pharynx, and quickly twisted and pulled, the violent hemorrhage being controlled by tampon previously prepared.

There is one point about the last case operated upon that I am watching with interest. Some German operators claim that if we destroy the larger part of the growth that the remainder is absorbed. I did not get all of the growth in the last case (a piece as large as a grape remained), as I had to stop on account of the condition of the patient. Transfusion of normal saline solution was used with gratifying results.

In regard to the operation, if the growth has a pedicle around which a loop of heavy wire can be passed, this should be done, and after using all force possible (without breaking the instrument) in tightening the loop and strangulating the growth, by twisting and pushing and pulling, the growth is separated from its attachment and removed. This can be done where the fibroma is pedunculated and the snare can be used, while the hemorrhage is very free for a few seconds, the tampon is not always necessary.

In cases like the last ones referred to, where size, shape and location or attachment did not permit of use of wire loop, the following method was used:

(1.) Parts cleansed as well as possible with syringe anteriorly and post-nasal.

(2.) Anaesthetic administered.

(3.) Tape pushed through nostril and brought out of mouth, to which was attached firm gauze tampon; this given to assistant to hold.

- (4.) Mouth gag securely inserted and held in place.
- (5.) Insertion of each blade of forceps, as you would obstetric forceps, being guided by the finger so as to engage largest and most important portion of the attachment as high up as possible.
- (6.) Blades are locked and patient turned well onto side, and head brought to edge of table. Everything being in readiness to meet any possible danger—transfusion apparatus with normal salt solution, etc.
- (7.) Growth being firmly grasped, by a quick and rapid motion is twisted and pulled out. The terrific hemorrhage which follows the detachment of the base is quickly stopped by pulling the tampon well into the post-nares and the pharyngeal vault by means of tape previously introduced. This may be removed in twenty-four to thirty-six hours, according to indications.



Plate V. Case 3.

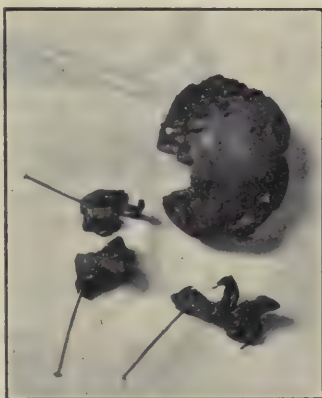


Plate VI. Case 4.

(Exact size.)

The two cases I have operated upon have almost proven true the predictions of Driffenbach, and have led me to devise and have made the forceps which I show you. The pattern is that of the adenoid forceps, the blades being much stronger, with a tooth edge specially constructed to prevent slipping. A lock similar to the obstetric forceps is used so as to facilitate easy and quick manipulation, the ratchet so as to firmly fix the handles and prevent the shifting of the blades.

Given another case like either of the ones reported, I would first ligate the external carotid artery of the side to which most of the growth was attached. The reports of Nicholson of Atlanta and Keene of Philadelphia have, in a number of cases, found no evil results from its ligation. On the contrary, both these surgeons

emphasize the simplicity and effectiveness of the operation. Not only in such cases would I tie this artery, but in any case where a severe hemorrhage followed or was likely to follow any operation within the nose or pharynx. The free anastomosis renders control of the main trunk devoid of danger to the life of the tissues, and assures the subsequent return of circulation to the extent of carrying on proper nutrition. This preliminary operation will make the use of the tampon unnecessary and prevent the so-called surgical shock. It is easily and quickly done, and I believe every throat and nose surgeon should be prepared to do it. With the hemorrhage controlled, the operation upon the two cases above reported would have been easily performed with the forceps I show you and not have been the bloodiest, most harrowing and uncertain of all surgical operations.



Plate VII.

Forceps for removing Fibroma.

In addition to my own cases, I want to show specimens from two similar cases operated upon by Dr. T. W. Moore, of Huntington, W. Va., with a brief history of each.

Case I. Perry McCoy, age 18. Tumor was first noticed four years ago. Patient claims to have suffered with intense headache all the time since he first noticed growth. At one time a prong protruded from left nostril, which was removed several months before I first saw him. Posterior portion of left nostril was entirely filled with tumor, which seemed to be wedged therein. Could pass finger about it in pharynx. After packing adrenalin solution about it (1-2000) I could move it, and ascertained that its only attachment was to posterior end of left inferior turbinate. After one hour's work I succeeded in passing a cautery wire over it. It was removed with difficulty after it was detached, and this started profuse hemorrhage that was controlled by packing posteriorly. The small growth was attached about the junction of the

middle and posterior thirds of the same turbinate. It was removed with the cautery snare after having broken No. 5 piano wire in steel snare. There was considerable pus flowing from the sphenoidal sinus, but patient would not wait to have it treated, and left for his mountain home (fifty miles of which he must walk) six days after the removal of the larger tumor, declaring that he felt better than he had for four years. Tumor weighed 240 grains. Operation performed December 3, 1902. Have no report from patient since he left on December 9, 1902.

Case II. Lewis, residence near Guyandotte, W. Va., age 12 years. Operation December 17, 1902. His family physician discovered growth one year ago. During this time he has been trying to absorb it by electrolysis. One prong protruded from left nostril, one edge could be seen below velum palati. Under chloroform I slipped a cold wire over tumor through mouth and removed by traction, it requiring considerable force. There was profuse hemorrhage for about five minutes, stopping spontaneously. Tumor was very firm, had but one attachment—the vault of the pharynx—and was decaying in two places, probably from pressure. After washing it weighed 14 drachms avoirdupois. The boy, who was weak and anemic, gained thirty pounds in six months, developing from an undersized, puny child into an extraordinarily large boy of his age.

Following is report of examination made by Dr. D. Brayden Kyle of Philadelphia:

1517 Walnut street, PHILADELPHIA, March 28, 1903.

DEAR DR. STUCKY—

Enclosed please find report on specimens which you sent me. It is most unusual to find four cases so nearly alike. I would be glad to have a reprint of your article. Your friend,

(Dictated.)

D. BRAYDEN KYLE.

To Dr. J. A. Stucky, Lexington, Ky.

REPORT OF SPECIMENS FROM DR. J. A. STUCKY.

Specimen No. I.—Sections from this specimen showed certain areas of fibrous tissue resembling very much in character the fibrous tissue due to inflammatory action, while other areas were of the soft variety, showing spindle cells and fibrillæ, and was exceedingly vascular. The blood vessels had very thin walls, but were especially numerous. Owing to the difference in the structure of the tumor it reacted rather curiously to the stain. I think, however, that instead of this peculiar reaction being due to any special cellular

change, it entirely depended on the fact that the more dense fibrous tissue took up the stain much more slowly than the softer areas. While the distinct fibrous areas were very small and not at all numerous, it gave to the section a rather odd appearance. At no point was there any evidence of malignancy. There were, however, a few areas in which the tissue was infiltrated with small round cells, but from the appearance of the surrounding tissue and of the blood vessels I am quite certain this was only a small area of inflammatory infiltration, and was not sarcomatous. I would scarcely call the tumor an angio-fibroma, but simply a highly vascular fibroma, as there is a distinct difference between a highly vascular tumor and angioma.

Specimen No. II.—Examination of this specimen showed almost the same appearance as No. 1. This is especially true of the connective tissue. However, the tumor was much more vascular, the vessels being very small and numerous. The walls of the blood vessels were fairly well formed and the surrounding tissue showed no signs of malignancy. I would give the same diagnosis from the microscopic appearance as in the preceding specimen, that of vascular fibroma. It is most unusual to find two tumors resembling each other so closely in microscopic appearance.

Specimen No. III.—This specimen has more the appearance of the myxo-fibroma. Certain areas, however, showed broken down tissue, which from its appearance, and the appearance of the tissue surrounding the area, I believe to have been an area of hemorrhage followed by necrosis. Some areas in which necrosis had not taken place supports this theory. There was, however, near the surface of this tumor, certain areas of small, round cell infiltration resembling very much, areas seen in the beginning of small, round cell sarcoma. However, I am inclined to think that it was nothing more than an inflammatory area, as in no instance did I find any blood vessels similar to those found in the sarcoma. While the tumor was fairly vascular, it was not markedly so as in No. 1 and No. 2.

Specimen No. IV.—This was almost identical with No. 3, with the exception that the epithelial layer was slightly thickened and some epithelial cells were found beneath the basement membrane, giving a suspicion at least of epitheliomatous infiltration. While the tumor was highly vascular, there was no marked alteration in the blood vessel walls.

DISCUSSION.

DR. JOS. BECK.—Last year at the meeting of the Middle Section of the Am. Med. Ass'n, I reported in conjunction with Dr. Holmes three cases of fibroma of the nose which were diagnosed as sarcoma, although only one proved to be such. The operation was done, not through the nose, but through the superior maxilla. One case was not ligated and that was reported as dead shortly after operation. In most of the cases reported by Holmes and Loeb the pedicle of these tumors were near the sphenoid bone. The two cases I reported were nearer the middle of the nose and consequently could not be operated on by the route described by the writer. It was necessary to remove the maxillary bone and empty the antrum, which was filled with fibrous masses. This year I had the pleasure of having a case which was diagnosed as a mucous polypus which could be seen in the post-nasal space, and its firm attachments led me to believe it was more than a myxoma. The trouble in the operation was in the engaging of a snare, and the doctor decided to take a heavy wire and with the help of the finger post-nasally pass it around and then thread it. It was the size of the doctor's second case, one of the largest masses I ever saw. Histologically it proved to be a fibroma. The bleeding after the operation was not marked.

DR. BARNHILL.—I wish to exhibit this specimen of a fibroid removed from the naso-pharynx of a man twenty years of age, for which a specially constructed steel snare was used, with canula large enough to admit a No. 7 steel wire. After four hours effort in tightening the loop, much to my chagrin, the canula doubled and the wire broke, rendering it necessary to abandon the operation or devise a new method. I pulled the canula away from the wires and re-threaded them into that of the Peter's tonsil snare, which proved entirely sufficient and severed the large mass much to my gratification.

I have seen several cases of naso-pharyngeal fibroma in the past few years, two of which I considered inoperable. The first, a boy ten years old was seen four years ago. The growth completely filled the naso-pharyngeal space, crowding the palate forward, immobilizing it, and sending a branch into one nostril of sufficient size to completely block it, and deform the cheek by its pressure. I have seen the boy recently and noted that he is no worse in any way, the size of the fibroid remaining about the same, and the boy's health fairly good. The late Dr. W. V. Morgan, an eminent surgeon of this city advised against an operation.

The second case was of a man about 30 years of age, in whom I believed the growth had been changed to one of malignant nature by frequent electro-cautery puncture, done for the purpose of reducing the growth. This case died of inanition, the size of the growth and the glandular infiltration causing final inability to swallow anything.

C. L. MINOR (Springfield).—I have at the present time a case of nasal fibroma springing from the posterior end of the middle turbinal. I can appreciate the difficulties that the gentlemen have had in removing them for I have broken a No. 6 piano wire on each of four different occasions in trying to remove the growth with a cold wire snare. The growth is as hard as a rock and can not be removed by any ordinary methods usually employed in nasal operations. Dr. Shurly advised me to loop the point of a galvano-cautery around the base and remove it in that way. I hope that he will relate to the society the method that he advised me to pursue.

DR. W. H. PETERS (Lafayette, Ind.).—I have removed a fibroma during the past year with my nasal snare. I applied the snare through the nose, over as large a portion of the growth as possible to get in the loop. The hemorrhage was not great. The case was a boy of 14. I used cocain and the operation required three sittings during a period of about an hour and a half. I have plenty of muscle, but it required all the strength of both hands to get through the growth. The growth had its origin in the nasal passages, and filled the whole of the naso-pharynx. In growths such as Dr. Stucky reports, however, no snare could be used, as it would be impossible to apply it.

DR. HAL FOSTER (Kansas City, Mo.).—I wish to congratulate Dr. Stucky on the success of his operations and the beautiful forceps he has devised. I have had some experience with extensive growths. Dr. Murphy was in the office and saw this patient and published the case in a journal. It was very large. I would vouch for what he says about snares, but the modification of Peter's snare seems to me meets the indication if you can get around the growth. If it can possibly be avoided, I think it is much better not to give these cases chloroform, because when you do they are much more apt to bleed.

I think the laryngologist should be prepared to meet any emergency that arises. Frequently you will have to do transfusion if you are not careful. The hemorrhage comes with a gush and is certainly serious. I used adrenalin; had a considerable hemorrhage but controlled it. I used the snare and then the forceps as the obstetricians do, and tamponed the nose very rapidly, with good results. I approach these cases with great anxiety and feel greatly relieved when the growth is removed.

DR. J. P. MORRELL (Terre Haute).—I am glad of the opportunity of reporting a case of which the account given by the doctor of his first case is a fair description. The patient was 17 years of age. The growth completely filled the right naris, and could be seen by raising the tip of the nose. A change in the contour of the bridge of the nose was also apparent. Upon elevating the palate the growth could be seen behind. I had had no experience in the removal of such growths. The removal of the growth had been previously attempted by another physician by whom two or three wires had been broken, and whose efforts had been followed by such violent hemorrhage that he desisted from further attempts. My attempt to remove a portion of it with a cold snare was rewarded with a broken wire. At most but a small portion of its anterior surface could be engaged in a snare. The growth so filled the cavity that it was impossible to surround any considerable portion of it by wire. I therefore adopted this method; With an electric knife I made a vertical channel into the growth and crossed this by a horizontal one. This divided the growth into fragments around which I could place the soft wire of the electric snare. By repeating this I was able in two or three sittings to reach the plane of the posterior edge of the vomer. I here found that the growth was adherent to the upper surface of the palate which effectually prevented my engaging it in the loop of the snare. I, however, succeeded in insinuating a wire with its end protected between the growth and the outer wall of the naso-pharynx into the oro-pharynx where I grasped it by forceps and pulled it out of the mouth. I was also successful in passing another wire with some difficulty around behind the vomer and to the opposite side of the growth into the oro-pharynx. Pulling this also out of the mouth the ends of the two wires were united. Traction upon the nasal end of one of the wires drew the wire loop thus formed out of the nostril until the junction was reached when the two ends were threaded in the snare, and traction made upon the growth. Then graduated traction was made extending over two days when the attachment was severed, and the growth removed. I have since then repeated the channeling process in removal of growths that entirely filled the nares, and consider it a very practical and ready means of getting rid of what would otherwise be a very troublesome condition.

DR SHURLY.—It is unusual to get the complications Dr. Stucky has spoken of. It is usual in operating to first destroy the adhesions, however long that may take, and subsequently arrange for the operation of its removal. In one instance I adopted the method of removing it piecemeal. This is not always as satisfactory. The cold snare is also quite unsatisfactory, although I have removed

them with the cold snare, by having the snare made of extraordinarily heavy wire and using No. 8 or No. 12 wire, although sometimes No. 18 will do better. The smaller the wire that will hold, the easier, because the smaller wire will cut through more readily. With the ordinary cold snare I found it almost impossible, without some additional guide, to keep the loop around the tumor. I pass the platinum wire through the nose; pass the fingers into the nasopharynx about the tumor, and gradually put the wire up behind the tumor with the fingers, or a probe which is to be bent. In that way, by threading the wire up over the tumor, I get the canula up to it. I then turn on the current, and by gentle traction cut through and remove the tumor. I have never met with these terrible complications, nor have I met with a tumor as large as the doctor's unless it was of a sarcomatous nature, in which case, the only way to do, perhaps, is to operate from the outside, cutting away the bone and all tissues as far as possible, in order to remove the whole sarcoma. I have never had success in removing them by the internal method when large, because I was never able to get all the growth out. A re-growth in most cases takes place. In growths of such a nature it would be very unsatisfactory to leave any of it.

DR. STUCKY (closing).—I appreciate the liberal discussion. In Dr. Beck's remarks, there is one point we must distinctly make, and that is the difference between a mixed fibroma and a true fibroma. I am sorry I did not have time to read all the reports and make this clear.

In answer to Dr. Barnhill, would say that I believe the electrocautery in these cases does harm and that by its use you stimulate the growth. I am sure it was true of the first case I had.

As to the remarks of Drs. Minor and Shurly regarding the attachment to the posterior turbinal, it is possible when located at this point to snare them, bone and all. But one point I tried to emphasize about this case was that the attachment involved the whole of the basilar process and extended up into the nose. You can no more make a loop of any kind to snare that than you can snare this piece of marble.

About the case described by Dr. Peters I do not see how with a snare of any kind I could have cut off this growth; and replying also to Dr. Foster, if I had made a little opening and attempted to channel through this growth, the patient would have bled to death.

About the adhesions, there is a difference between the adhesions and the attachment. I broke up the adhesions gradually and kept them separated with strips of gauze. The main attachment was on the basilar process.

AN UNUSUAL CASE OF SPONTANEOUS, BILATERAL HEMORRHAGE FROM THE EAR.

BY M. A. GOLDSTEIN, M.D., ST. LOUIS, MO.

While there is but scant literature available in the question of hemorrhage from the ear with an apparently normal condition of the areas under examination and an intact external auditory canal and drum membrane, the data furnished in the reported cases of Ferreri,¹ Stépanow,² Eitelberg,³ Gradenigo,⁴ Stein,⁵ Richardson,⁶ and Wheelock,⁷ establishes beyond question the fact that this phenomenon occurs. From a detailed study of these reported cases, I gather that all but one were in females; that five were women of hysterical temperament; four were vicarious to menstruation, and in all but one, the hemorrhage was intermittent in character and infrequent. In the cases of Gradenigo and Richardson there was a distinct syphilitic history.

In all of the cases thus far reported, the hemorrhage from the ear occurred at irregular intervals, and in all but one (Stein), the bleeding was unilateral. In the cases of Ferreri and Stepanow the discharge was profuse, lasted several days, and the quantity of blood exceeded that of an ordinary menstruation. In the cases of Eitelberg, Gradenigo, and Wheelock the quantity of hemorrhage was small and of short duration. As the case which I report has some data analogous to this literature, I have incorporated herewith a synopsis of all the cases thus far reported pertaining to hemorrhage from the ear, associated with intact membrana tympani. A careful comparison of these data with my own case reveals interesting points of resemblance, and many points of difference.

At the outset I wish to distinctly establish two facts in the etiology of my case. First, that the question of simulation or malingery is definitely excluded, as the patient was under careful observation for over one year, and as the corroboration of competent observers will testify. Second, that the menstrual function did not influence either the quantity or the time of this hemorrhage.

Notwithstanding the fact that the patient was under my personal, frequent and careful observation for more than one year, the data which I am prepared to offer are scarcely sufficient to establish definitely either the etiology, pathology or diagnosis of this case,

and I may be pardoned for adding many points which ordinarily might appear superfluous.

Miss X., white, age 22 years, intelligent and of good family, one of twins, of distinct hysterical temperament, born in Cincinnati. Her family history is good; it is stated that up to her sixteenth year her health was perfect. For data of her first illness I am indebted to Dr. J. M. Pace, of Dallas, Texas, which I can summarize as follows: The patient applied September 25, 1897, for treatment of a lacerated gum in the lower maxilla, following the extraction of a tooth. A spicula of necrotic bone was removed from this infected area, and paroxysms of pain, hysteroid or tetanoid in character continued. In a consultation one week later, tetanus was suspected, but nothing of that character developed. The hysteroid condition continued, some days better, then again worse, with sleepless nights, until October 28th, when improvement began. The acute stage of this illness, therefore, lasted about one month. During this illness her temperature varied but slightly from normal. The wound discharged a sanguinous, purulent, sanious, offensive fluid. Some necrosis of gum and bone was noted. She continued in a nervous state for some time after recovery; in fact, until the family moved to St. Louis in 1898.

The feature which was most pronounced in this attack and which I desire to emphasize, is the hysterical temperament observed in the patient,

In September, 1901, she first came under the observation of Dr. J. Friedman, of St. Louis, to whose courtesy I am indebted for this case, who has furnished me with data of her last illness, and who has carefully watched with me the many interesting developments during the past year.

DR. FRIEDMAN states:

The history of the case dates some three or four years back. While at a summer resort patient was shocked by an electric bolt, which shock was followed by an hysterical attack. This attack did not differ materially from other attacks of hysteria with the exception that she had pain in the right inguinal region in the neighborhood of McBurney's point. No temperature and no increased pulse rate. The January following this attack patient again had extreme pain at McBurney's point and while the muscles were not tense and patient would admit of a great deal of pressure, this pain was accompanied by incessant vomiting. Patient would absolutely retain nothing on her stomach, but would vomit everything taken. The only food that patient could retain was raw ripe pears. Again she had no temperature, and diagnosis was hysterical vomiting. This attack continued some six (6) weeks, finally yielding to a nerve treatment.

In February of last year (1902) she again had a similar attack, again accompanied by incessant vomiting, and on the recovery from this attack she called my attention to the fact that she was bleeding from both ears. She complained of considerable pain, which pain was not apparent before the bleeding. I referred her to Dr. Goldstein, who from that time until her recovery treated her. I wish to mention the fact that the left ear yielded to the doctor's treatment in a very short time. The right ear continued to show evidences of bleeding for eleven months. There were repeated bacteriological examinations of the fluid coming from the ear, and it was pronounced to be "blood and serum."

J. FRIEDMAN, M. D.

On March 9, 1902, I was first called to see Miss X. She was convalescing from this last attack, and was sitting up for the first time. Several hours before I reached the patient there had been a spontaneous, bloody discharge from both ears, coming on without any subjective symptoms beyond a slight fullness in the head. There was no previous history of trauma, nor had there been an ear trouble of any kind. I would emphasize that it was definitely determined that the hearing was normal prior to this attack. On examination, I found both external auditory canals filled with a sero-sanguineous fluid, which on wiping away left no stain nor did it clot. This fluid indicated slightly alkaline reaction with litmus paper. On wiping away the discharge and clearing the auditory canal, I was surprised to find the membrana tympani intact. Both membrana tympani were in normal plane, no part of their surface showing either bulging or retraction, and there was absolutely no evidence, by inspection, of any perforation. There was no evidence whatever of inflammation, nor has there been any during more than one year since I have had the case under observation. The only clinical data which I can report is a sense of pressure and occasional headache just before the exudation took place. The discharge occurred at irregular intervals; earlier in the course of the affection, as often as every half hour, when the patient was under some nervous excitement, there was a greater frequency and quantity of the discharge. The quantity of each exudation was of about the same volume (about one c. c.). The discharge occurred more frequently by night than by day; the recumbent position seemed to favor it. Four days after I first saw the patient, the discharge in the left ear ceased, and there has never been a recurrence in that ear; the other ear continued to discharge for fifty (50) weeks without an intermission of more than eight hours. The character of the exudate is best described in the bacteriological report which is herewith appended:

ST. LOUIS, June 18th, 1902.

Dear Doctor:—I have to make following report of my analysis of fluid from ear of Miss X.

Reddish colored fluid, of offensive odor. Specific gravity 1040, determined after Hammerschlag's method, with equal parts of chloroform and benzol.

Gave none of the fibrin reactions. Fluid was alkaline in reaction. Hematoidin present. Teichmann's hematin crystals present. Distinct albumin reaction present. Testing with cupric solutions failed to give any reduction. 2 c. c. of this fluid was put into an Einhorn saccharometer tube, with yeast and distilled water; no gas formation after 24 hours.

Microscopic examination of sediment obtained by centrifugalization showed a few red blood cells and many polymorphonuclear leucocytes. No lymphoid cells or lymphocytes were made out.

A great many short, motile rods were present. I did not make any attempt to identify them, because the vessel in which the fluid was stored was not sterilized, so these bacteria might have been a contamination from the bottle.

I conclude that this fluid is partly blood. The main constituent is a serous fluid of some kind. Whether it is cerebro-spinal fluid cannot positively be stated. We have no positive test for cerebro-spinal fluid. Cerebro-spinal fluid is generally very poor in albumins; this fluid gave marked albuminous reaction. Cerebro-spinal fluid does not clot nor does it give fibrin reaction; this fluid corresponds to the behavior of the cerebro-spinal fluid in that respect. Cerebro-spinal fluid occasionally contains a substance which like dextrose, reduces Fehling's solution, but which is not sugar; this fluid did not reduce, nor did it give a saccharine reaction with yeast in the fermentation tube.

What is known as to the composition of cerebro-spinal fluid relates chiefly to *normal* fluid; the fluid in this case may be altered cerebro-spinal fluid, blood-tinged, hence the albuminous reaction might be accounted for in that way.

Very truly yours,

R. B. H. GRADWOHL, M. D.

The examination of the blood of the patient added below, shows no dyscrasia:

ST. LOUIS, July 18th, 1902.

Dear Doctor:—I beg to submit herewith report on examination of blood of Miss X.

Smears from blood obtained from lobe of left ear, fixed with equal parts of alcohol and ether, stained with eosin and methylene blue, with Jenner's stain and with polychrome methylene blue shows pale red blood corpuscles, with marked poikilocytosis, many megaloblasts being present, besides a number of blood plaques. No morphologic, pathologic change in the white blood corpuscles was to be noted. No parasites were noted in these examinations. Blood counts: Red, 3,000,000; white, 5,000.

Very sincerely yours,

R. B. H. GRADWOHL, M. D.

Hearing tests made a few days after my first examination of the patient revealed the following: Conversation voice readily heard by the patient. Forty inch watch not heard on contact on either ear. Tuning forks C_1 - C_2 - C_3 , and C_4 not heard over mastoid on either ear.

Tuning fork tests made one week later (after discharge had ceased in left ear) showed slight perception over left mastoid; Weber's test also elicited tone perception in left ear.

Tuning fork tests made December 10, 1902, indicated Rinne positive and the watch test $\frac{3}{4}$ on left ear. Membrana tympani at this time appear normal on inspection.

On determining the source of this sanguineous exudation, I had constantly in mind the observations made in the cases previously reported of a similar character. In all of these cases the origin of bleeding seems to have been from the cerumen glands, especially those nearest the fundus of the auditory canal.

While I have never been able to definitely locate the exact point at which this sanguineous fluid made its appearance in the ear, I have always located it in the angle formed by the posterior wall of the auditory canal and the plane of the membrana tympani. I have watched the auditory canal and membrana tympani by well reflected light for twenty minutes constantly, in the hope that I could determine the location of the exudate; I have seen the exudation suddenly fill the fundus of the canal on several occasions, but I could not locate the bleeding points.

As the case progressed, the quantity of the discharge increased. The patient went about with large pads of cotton over the ear, and these were soaked and often dripping with this discharge. I sought consultation locally, and presented a synopsis and data of the case to distinguished confreres abroad, and I have taken the liberty of recording in brief their opinions and suggestions of the case.

I even went so far as to do an exploratory operation May 20th, to examine and determine the character of the cavum tympanum, cutting a fenestra in the membrana tympani, and packing the middle ear cavity with styptic gauze. Even this exploratory examination gave us no new data.

The patient spent the summer months at the lakeside in Wisconsin, and in spite of the uninterrupted bleeding from the ear, gained fifteen pounds in the course of two months.

The diversity of opinions of many of my consultants was so marked, that I was undetermined what course to pursue in the further conduct of the case. I felt assured that this prolonged exudation carried with it no serious lesion, for never during the course of the case was there the slightest evidence of inflammation, rise in temperature, or serious symptoms. Had the case been associated with any cerebral lesion, there certainly would have been definite

development in short order. By exclusion and elimination, the only etiological factors which were still subject to consideration were, *a.* malingering, *b.* neurosis. The definite exclusion of malingering or the introduction of a fluid extraneously has been established by my careful observations and the corroboration of my consultants.

At different stages of its development, this case was seen by Drs. J. Friedman, J. B. Shapleigh, C. A. Todd, J. C. Buckwalter, W. E. Klokke and H. W. Loeb, of St. Louis; J. M. Pace, of Dallas, Texas, and H. V. Würdemann, of Milwaukee, Wis. I add in brief the opinions expressed by some of my consultants as to the etiology and character of this case.

Sr. Louis, April 6th, 1903.

Dear Doctor:—In regard to the case of Miss X., and especially as to the possibility of malingering about which there has been much discussion, I have this to say: Malingering is the easiest explanation of the case, but I cannot accept it for two reasons. First, that you yourself have seen the fluid suddenly well up from the depth of the auditory canal on several occasions while carefully wiping the ear; second, that on one occasion the bleeding began while the patient was sitting quietly in my office chair. I do not see how the fluid could have been introduced from without in this instance.

There are many other things against this supposition. For instance, how could the patient have obtained a fluid of such peculiar character in such quantity, and under such a variety of circumstances during the past year?

I do not think this a case of malingering. What then, is the explanation? Was it labyrinthine or cerebral fluid? I think not, for there was too much blood in it under non-inflammatory conditions.

It was not a vicarious menstruation, for that function was performed normally, and with no effect on the ear trouble.

I have a theory which I advance with hesitation, and in anything but a dogmatic spirit. It is the best analysis of a most interesting, unusual and obscure pathological condition that I can make from what I have heard and seen of the case. The theory is, that this bleeding was a nervous phenomenon of the vaso-motor origin and that the fluid was by osmosis, or sweating through the capillary walls. In favor of such an hypothesis these facts may perhaps be advanced: First. The patient was of a distinctly nervous temperament, tending to the hysterical. This is very favorable to the appearance of nervous phenomena. Second. The deafness which still persists, is I believe hysterical. It certainly depends on some condition of the perceptive apparatus of the ear. Third. Disturbance of the capillary circulation in other regions. I noticed frequently such a disturbance of the capillary circulation as shown by red blotches in the skin of the cheek and neck—areas of vaso-motor paralysis. These were transient and would come and go during the period of an examination. Fourth. The character of the fluid and the manner in which it made its appearance together with the intermittent nature of the flow. The fluid was sanguinolent, and did not clot; it appeared quickly after a sense of fullness in the ear and no special point could ever be detected as to the origin of the hemorrhage. This is to me suggestive of an intermittent vaso-motor paralysis producing first, congestion of the capil-

laries and then a transudation through their walls of the serous elements of the blood with more or less of the red corpuscles. Fifth. The cessation of the phenomenon through mental suggestion. A supposed operation, carried out as you did this one would produce a decided mental effect in a semi-hysterical patient.

This theory does not answer the question what capillaries were at fault—those of the tympanum, membrana or auditory canal, and if the former, how did the fluid escape without rupture of the membrane, which should have been detected. If the oozing came from the canal, then of course no opening in the membrane need be accounted for. Why did it persist in this ear and cease in the other? This is less important, for often no reason can be given for the localizing of hysterical phenomena in certain regions.

The case certainly falls outside the usual lines in its symptoms, course and etiology and the theory I have advanced seems to me to cover the ground better than any I can think of except malingering, and this I have said I cannot accept.

Yours truly,

J. B. SHAPLEIGH, M. D.

Among the therapeutic measures which were brought into use, were included the Violet-Ray Lamp and the X-Ray, and I add the following data of X-ray treatment:

ST. LOUIS, March 21st, 1903.

Dear Doctor:—I am pleased to furnish the following data in the case of Miss X., and my observation in the X-Ray treatment to which she was subjected.

The patient was brought to me December 5th, 1902. She had hemicrania, especially right side; seemed languid, face slightly flushed, skin irregularly mottled, hands cold, restless and of nervous temperament.

She bears static current in fine spray well; has anæsthetic zones (static interrupted) over areas illustrated. Area 1, anæsthesia absolute; area 2, anæsthesia relative.

First treatment; static interrupted current and vacuum electrode, eight minutes. Patient claimed at second sitting that hemorrhage was increased.

As no appreciable change was observed after four or five treatments with a static machine, this was discontinued and the X-ray applied.

External auditory canal was wiped dry revealing a clear picture of normal membrana tympani, without opacities. A lead aural speculum was closely fitted into the canal, the X-rays isolated by means of a cone of sheet lead and the rays of a medium hard tube at twelve-inch distance, allowed to enter for ten minutes. The patient complained of temporo-sphenoidal headache following the exposure.

At a subsequent use of the X-ray I made an inspection of the auditory canal and membrana tympani after the tube was removed, and after patiently waiting some ten minutes was rewarded by seeing a quantity of sanguinous fluid suddenly well up from the fundus of the canal. This fact convinced me absolutely that the question of simulation or malingering which had been suspected and much discussed, was no longer in debate in this case.

I suspected this to be some form of vaso-motor neurosis, and would refer to the works of Schwalbe and Vierordt where mention is made of the hemorrhage of hysteria from the nose, throat, stomach, bowels and skin. I think the etiology of this case will be found along these lines.

Yours very truly,

W. EMIL KLOCKE, M. D.

During the summer while the patient was in Wisconsin, I had occasion to refer her to Dr. H. V. Würdemann, of Milwaukee, Wis., and append abstract of his letter :

MILWAUKEE, WIS., September 8th, 1902.

Dear Doctor:—The case of Miss X. is extremely interesting. Without further data from you, my opinion expressed in the following is made up entirely from the one observation of the case that I have made.

I found no temperature; total deafness of the right ear; left practically normal; an intermittent serous discharge (which the microscope shows to contain many red blood corpuscles and a few epithelial flakes) gushes from time to time out of the external meatus in sufficient quantity to soak thick external dressings; it is worse in a reclining posture. There is some incoördination on walking with eyes closed. The previous history of vomiting and vertigo probably have reference to the internal ear. There is a small perforation posterior to the end of the malleus (this perforation resulted from the exploratory operation made May 20th, 1902).

I do not think the secretion is made in the middle ear, but that it comes from the cochlea and is probably connected with the cerebral cavity, I think however, that the red blood corpuscles and epithelium which are in the fluid are probably more or less from the middle ear.

The case is certainly very uncommon, but to my mind, is of the same character as those occasional cases we see in which a discharge of this nature occurs for some hours or days after fracture of the skull, in which the fracture extends through the petrous portion of the temporal bone, allowing the cerebro-spinal fluid to leak out through the middle ear. I am greatly obliged to you for the reference of this interesting case.

Cordially yours,

H. V. WÜRDEMAN, M.D.

In my correspondence with the eminent authorities abroad, Prof. Politzer of Vienna, Prof. Lucae of Berlin, and Prof. Urbantschitsch of Vienna, it is worthy of remark that there was a consensus of opinion expressed as to the etiology of this case. I had furnished to each of these authorities a brief report of the clinical data which had been gathered, including a description of the sanguinolent fluid, its quantity and analysis, the method in which it suddenly welled up in the fundus of the canal, the tuning fork tests, and all other information of a positive character that I could obtain. From this rather incomplete report, each of the above authorities independently diagnosed the case as one of simulation. Prof. Urbantschitsch added as another possible cause, that of trophic neurosis.

In the light of these and similar suggestions by local consultants as to the case being one of possible malingering or simulation, I proceeded to systematically and absolutely eliminate this etiological factor. My evidences for excluding malingery are: First, the most careful inspection of the auditory canal and tympanic mem-

brane failed to reveal any evidence of the parts having been tampered with. Second, the very character and constituency of the fluid was proof against it. The fluid was not blood, nor did it coagulate. Third, on at least six occasions, after having wiped the canal dry and while patiently inspecting the affected area, I have seen this fluid suddenly well up in the fundus of the canal. One of the unusual features associated with the determination of the source of this bleeding, is that I was unable to locate the exact point from which the fluid entered the auditory canal. As I had definitely in mind the distal ceruminous glands as a possible source of this exudation, my inspection always included this area and I can say positively that the fluid welled up from points farther remote than the most distal ceruminous glands. On wiping away the exudate (usually about 1 c. c. in volume), I could determine a moist, reddish, glistening line along the posterior circumference of the membrana tympani, extending from a line with the short process of the malleus, to the floor of the canal. This line always appeared about the same length, and in the same position at each inspection. Fourth, I do not offer my own testimony alone as to the sudden welling up of this sero-sanguinolent exudate in the ear, but submit as corroborative evidence the observations of Drs. Todd, Shapleigh, Buckwalter, Klokke, and Würdemann, all of whom saw this exudation at different times. Fifth, as an additional measure to exclude simulation, I proceeded to seal the affected ear in the following manner. The external auditory canal was thoroughly cleaned and dried, and then lightly filled with sterilized cotton which had been previously colored blue, and which was inserted into the ear without the patient's knowledge as to the change in color of the cotton. A small tampon of white sterilized cotton was placed over this, the entire concha filled with cotton, a piece of gauze shaped to the concha, and the whole dressing fixed with collodion. This dressing was retained in position for twenty-four hours. When removed, all of the dressings were still in the position in which they had been arranged, and were saturated with the exudate.

I think I have furnished sufficient evidence to absolutely exclude simulation or malingering from the etiology of this case.

In the entire literature which I have investigated, there are but two other factors associated with some form of neurosis which have been mentioned. One is that of vicarious menstruation in hysterical female subjects; the other is the case of Stein of Moscow, of a young boy with hæmophilic diathesis. Neither of these conditions are applicable to my case. In all of the previous cases reported, the source of the exudation was either definitely determined or supposed

to be from the ceruminous glands; in my case, careful, repeated observation proved to me that the ceruminous glands were not the source of the exudation. In all of the other cases the exudation consisted of pure blood; in my case the fluid (as indicated by chemical and microscopic analysis), was not pure blood, nor did it have the usual properties of blood.

By this process of elimination, I have been able to exclude all of the previously known causes which have had any bearing on similar cases, but after one year's careful observation I am unable to add any further positive data concerning the etiology of this case.

After taking every precaution and attempting every therapy, I finally determined on radical suggestive therapy. February 22, 1903, the patient was taken to the Jewish Hospital of St. Louis, and both Dr. Friedman (her attending physician), and I positively declared to her that an operation would be performed on the affected ear, that the entire head would be encased in a fixed plaster of Paris dressing, and that the operation would absolutely and permanently cure the ear. This statement was firmly impressed on the patient by several repetitions.

Chloroform to primary anaesthesia was administered, the auditory canal was dried out and lightly packed with a narrow strip of gauze and with a small pledget of cotton, the entire head enveloped in sheet wadding and a heavy plaster of Paris bandage applied, enveloping the head and neck. The patient was kept in a recumbent dorsal position and carefully watched for forty-eight hours. The fixed dressings were then cut away, and when the gauze was removed from the affected ear there was no evidence whatever of a recurrence of the exudate.

A stiff crinoline bandage was then applied for forty-eight hours, and renewed daily for one week. At the end of that time the dressings were removed entirely and the patient pronounced cured. There has been no sign of recurrence of this exudation to date (a period of six weeks).

Another corroborative evidence of the hysterical nature of this neurosis is the present condition of the hearing. Throughout the period of exudation, there was total deafness in the affected ear; to-day there is Rinne positive and the hearing for the watch 20-40.

The case certainly has no parallel in otological literature.

ABSTRACTS OF ALL CASES OF HEMORRHAGE FROM EAR WITH INTACT MEMBRANA TYMPANI.

Alcani cenui eziologica sull' emorragie dell' organo uditivo e descrizione d'una otorragia isterica. (The etiology of hemorrhage from the ear, with report of a case of hysterical otorrhagia.) G. FERRERI, (Turin.) *Sperimentale*, 1882, *Maggio*; *Archiv. f. Ohrenheilkunde*, 1883, Vol. XIX., p. 173.

The author cites in a general way the various etiological factors on which hemorrhage from the ear may be dependent, and then describes the following case:

Female, 30 years old, single. Has three sisters without hysterical tendencies. Menstruation began at 12 years, and until five years ago was always regular. Even as a young girl she had frequent rhinorrhagia, and later this became a regular feature either with the beginning or cessation of the menstrual flow. Six years ago, as the result of a severe fright, there was amenorrhœa for three months and there appeared at regular intervals short, convulsive, nervous phenomena. This nervous condition then became serious in character and the patient was confined to bed for eighteen days in an almost lifeless state; speech and consciousness then returned. She was bedridden for six months and under constant treatment.

Whenever her menses were due the patient had extreme pains in the left ear and in the left side of the head, with slight bleeding from the ear. Examination by de Rossi at this period revealed an extreme sensitiveness of the ear, intense pain with every movement of the jaw and a severe otorrhagia so profuse that the patient called on de Rossi at 5 a. m., who used collodion to check the bleeding, as it was the nearest remedy at hand.

The patient was then placed under observation in the ear department of the hospital for two months. She showed many hysterical tendencies, melancholy temperament and lymphatic constitution.

For several days after this severe hemorrhage the left cheek and auricle were swollen and red, and there was hyperæsthesia of the n. acusticus, and pain in the left side of the head. Examination of the ear revealed only a normal status, and the cause of the bleeding could not be determined. She was given emmenagogue and tonic treatment. After two months she again menstruated. On several occasions while still in the hospital and closely examined, de Rossi determined the exudation of blood from four or five openings of cerumen glands in the postero-inferior area of the external auditory canal, and but few m.m. distant from the auditory meatus. The blood appeared rapidly, drop by drop, so that in several hours two or three handkerchiefs were stained with it. This otorrhagia recurred at irregular intervals, and of varying volume. Careful observation of the vascular system indicated evidences of Basedow's disease.

Five months after her discharge from the hospital there was a material improvement in her condition. Menstruation was normal, there was no otorrhagia and no hysterical phenomena. Two years later patient stuck herself accidentally with a long needle, deeply penetrating the third metacarpal space and a portion of the needle was broken off in the tissue. Then followed various hysterical conditions plus a permanent contraction of the arm on the injured side. The wound healed, menstruation remained normal and there was no further otorrhagia. Later there followed periostitis acuta, of

the upper third left humerus, and an incision eight c.m. long was made, and a fragment of the needle extracted. After the operation the hysterical phenomena were again accentuated by absence of menses and bleeding from the nose. Later there was frequent cephalalgia left side, and hemiplegia, with entire loss of motion and sensibility. The hemiplegia improved to such an extent that patient was able soon to use the left arm. Dysmenorrhea persisted irregularly and there was frequent bleeding from the nose and twice there was moderate, and once a profuse, bleeding from the ear.

As the improvement of the hemiplegia was first observed in the upper extremity, the author records this case as one of vaso-motor disturbance rather than that of a possible lesion in the central nervous system.

Vicariirende Ohrenblutungen mit voruebergehender Taubheit combinirt.
(**Vicarious Otorrhagia and Transitory Deafness.**) STEPANOW. *Monatsschrift f. Ohrenheilkunde*, No. 11, 1895.

The patient, a female, aged 17, came of healthy family and had no sickness up to her 13th year. First menstruation at 13. About two months before underwent a severe operation of extraction of needle from hand without narcosis, on account of which fell into a faint that lasted 24 hours. The first menstruation was accompanied by considerable disturbance on the part of the nervous system, which kept the patient in bed. After getting up, she had paralysis and anesthesia of both legs. After some months the paralysis gradually disappeared; the anesthesia remained longer and disappeared suddenly. Since then the patient did not menstruate. Instead, there occurred from time to time hemorrhages, at first out of both, but late ralmost exclusively out of left ear. They lasted 1 to 2 days, and were accompanied by dyspnea, cardiac palpitation and pain in region of heart. Sometimes these disturbances occurred also between the ear hemorrhages.

During the year past, the bleedings have grown worse and have appeared in almost regular monthly periods:

Examination of the ears showed:

Right. Hearing is normal. Membrana tympani exhibited only a slight opacity in middle. Manubrium and light reflex well marked.

Left. Hearing good, but less than on right side. Manubrium mallei does not stand out so prominently; processus brevis is well marked. A central opacity of membrana tympani.

In auditory canal no alterations evident. Bone conduction better on right side than left.

Naso-pharynx. Some chronic catarrh.

Pharynx. Chronic granular pharyngitis.

The hemorrhages were generally preceded for a longer or shorter time by prodromata; viz., a sharp lancinating pain, diminution of hearing on left side, with vertigo and general weakness.

The author had opportunity to see this patient in the prodromal stage, during the attack and after.

In the prodromal stage, he found considerable diminution of hearing of left ear; watch heard neither by air nor bone conduction.

The otoscopic examination showed no alteration. Eustachian tube proved to be patent. Hearing and subjective noises not influenced by inflation.

A hemorrhage took place twice early in the morning and, according to those present, it was copious and fully equal to that from a normal menstruation.

The author, after cleansing the ear, found, to his astonishment no change except slight maceration of the parts. There was even no sign of hyperemia. Mastoid was tender to the touch. Judging from catheterization tympanum was empty. No perforation rale. No trace of blood in nose or naso-pharynx.

Hearing in left ear is now completely gone, so far as could be judged with intact condition of other ear.

In order to determine whence blood came, author endeavored to examine ear during hemorrhage, but had the peculiar experience that, whenever, with speculum in hand, he waited for bleeding, it never came; whenever he desisted from examination, the canal would overflow with blood.

Author concludes that without doubt the blood came from the walls of the auditory canal and possibly also from membrana tympani, although the absence of blood in tympanum was opposed to latter idea. Second, that blood came from this side of membrana tympani and not from tympanum; and the bleeding was accompanied by complete anesthesia of the audiotry canal, vertigo and tinnitus.

As to the nature of the bleeding, can only say that it probably took place by diapedesis, and was, perhaps, analogous to the so-called stigmata. The sebaceous glands played some role.

Ein Fall von periodisch wiederkehrender Ohrenblutung bei imperforirtem Trommelfelle. (A Case of Periodically Recurring Otorrhagia with Imperforate Membrana Tympani.) EITELBERG. *Internat. Klin. Rundschau (Wien)*, 1898, II, 81.

Patient was an anemic, badly nourished woman, age 37 years. As a child often complained of pricking in the ears. In 1870 had suppurative otitis media in right ear, which lasted a long time and was accompanied by frequent hemorrhages from the ear.

Examination, however, reveals evidence of inflammation having at some time taken place also in left ear.

In 1874, patient was seized with intense occipital and parietal headache, which led often to fainting spells. This improved after patient was married. She had borne five children, of which, however, only one lived. All he rest died in about three years in convulsions. During pregnancy patient was free from headache and earache. Headache was said to be worse always at time of new moon.

Examination of ears gave following: On right membrana tympani there was posteriorly a scar, while almost entire lower half was occupied by a chalky deposit. On left membrana tympani could be seen two long oval scars in front and below a crescentic-shaped chalky deposit. In left ear there was intermittent tinnitus. In right ear no tinnitus, but often pain. There was chronic rhinitis with swelling of turbinates and Eustachian tube. On right side bone conduction was better than air conduction. Tuning fork on vertex could be heard only on right side, from teeth and over mastoid on both sides, though on right much better. Watch heard normally 6 m. could be heard on either side not above distance of 10 c. m. Right side, numbers spoken in whisper heard 1 meter. Left side 50 c. m.

The patient first came under writer's observation June 20, 1897.

Aural bleeding was always from left ear, although the right seemed most affected. In two months patient had two such severe attacks that she was

compelled to go to bed. Following prodromal symptoms were noticed: in left ear, intense heat sensation; there was sometimes also an itching in external canal, which was relieved by rubbing the tragus; there was diminution of hearing. Sometimes hemorrhages would come on during exertion; at other times when doing nothing.

Amount of blood lost usually one or two teaspoonfuls and hemorrhage is followed by disappearance of pain, if that was present. There is now noticed the following peculiarity in her symptoms, viz.: that in lying on the *right* ear an unendurable beating sound is heard in the *left* ear and vice versa.

Observation for some months demonstrated that there was undoubtedly a relationship between the aural hemorrhage and the menses. However, there did occur some hemorrhages outside of the time for menstruation and besides it occurred along with period on some occasions, but these were exceptional.

Author was able to exclude simulation and states that careful examination showed no trace of a past traumatism.

“A Case of Periodically Recurring Hemorrhage from the Ear with Imperforate Membrana Tympani, in Hysterical Patient.” G. GRADENIGO, *Archiv. f. Ohrenheilkunde*, Vol. 28, p. 83, 1889.

Female, age 15 years, hysterical temperament. At the age of fourteen large erythematous blotches were observed symmetrically and bilaterally about the tibio-tarsal area; ulceration followed, and cicatrization was very slow.

In consequence of a severe coryza, patient suffered with constant headache, daily exacerbations of severe pain in the frontal region, and frequent attacks of sneezing. These sneezing onsets were paroxysmal, and often of several days duration, so that the patient became exhausted thereby. As the sneezing attacks subsided, patient located pain in occipital area and complained frequently of severe bilateral earache.

Functional examination revealed impairment of hearing on both sides, accompanied by indefinite anæsthesia of the auditory nerve; chronic catarrhal otitis media bilateral; hypertrophy of the anterior end of the right lower turbinate; deflection of the septum to the left, and slight chronic catarrhal pharyngitis. Under suggestive treatment and Politzerization patient was temporarily improved, but there were soon recurrences of pain in the ears and occiput. About three months later there were recurrences of the sneezing paroxysms, and distinct hyperplasia of the mucosa of the naso-pharynx and pharynx. It should also be observed that a periauricular edema was also noted, and there was some dry cough and dysentery.

When this symptom complex was at its height, patient menstruated. The night previous to menstruation a few drops of blood exuded from the right ear. An examination on the following day failed to reveal any traces of this bleeding in the external auditory canal, and no morphologic change in the appearance of the drum membrane. The patient was carefully observed in subsequent menstruations, but no further bleeding from the ear was found; the pains in the occiput and ears, however, continued. Six months later the patient again bled from right ear. No distinct evidence of bleeding point could be found on examination excepting the presence of small flakes of dry blood adhering to the drum membrane and auditory canal. On the posterior wall of the bony canal, there were six to eight red punctate spots which the

author claims were the mouths of the cerumen glands, and states this as the possible origin of the bleeding. The case was successfully treated by the instillation of saturated solution of boracic acid in alcohol.

Ein Fall von Ohrenblutungen bei einem Knaben mit Imperforirtem Trommelfelle. (A Case of Otorrhagia with Imperforate Drum Membrane, in a 13 year old boy.) S. VON STEIN (MOSCOW.) *Zeitschrift f. Ohrenheilkunde*, 1893, Vol. XXIV., p. 294.

When the author was first called to this case, a thirteen-year-old boy had a sudden profuse hemorrhage from both ears, saturating two handkerchiefs and lasting about three hours. A careful inspection revealed in both auditory canals a number of punctate bleeding spots close to the junction of the concha and external auditory canal, seemingly the openings of well-developed cerumen glands. The rest of the external auditory canal appeared free of blood; the membrana tympani were normal, and with no evidence of hyperemia; repeated tests indicated normal hearing.

There was a moderate catarrhal rhinitis; the general condition of the patient was good; he complained neither of pain in the ear nor elsewhere, simply appearing anemic, which was his usual condition.

Since he was six years old he had had frequent attacks of epistaxis, especially in the summer time; in the three years just preceding the otorrhagia, this nasal hemorrhage became more severe and more frequent, usually accompanied by intense migraine. This nasal hemorrhage was especially aggravated when the patient engaged in gymnastic exercises, and on the day the otorrhagia occurred he had been quite active in the gymnasium.

The day following the bleeding, the author, on removing the tampons, noticed a few drops of blood exuding from the mouths of the cerumen glands. When these drops were wiped away, red punctate spots remained. The bleeding recurred for four consecutive days in gradually diminishing quantity. The membrana tympani and the hearing were always normal.

This hemorrhage tendency ceased as soon as the boy was cautioned against too strenuous exercise. In this case the author definitely determined that the blood exuded directly from the mouth of the cerumen glands.

Report of a Case of Hystero-epilepsy in which the Climax of the Seizure was expressed by Discharge of blood through the Intact External Auditory Canal. K. K. WHELOCK, (Fort Wayne), *American Medicine*, August 3, 1901.

Female, white, age 28. Four children, apparently in good health. Had history of previous treatment for general nervousness, attacks accompanied on several occasions by copious hemorrhages from ears alternating. Family history good, excepting father, who, for five years had attacks simulating epilepsy, now completely free from them.

Inspection showed normal canals and tympani. No history of trauma or suppuration from ear. Hearing diminished in both ears, membranes retracted slightly, but no perforations.

Two years before birth of last child she had fainting spells at menstrual periods, with loss of consciousness, tonic and clonic spasms, frothing at mouth, etc. Since birth of last child, these attacks ceased, but were replaced by periodical hemorrhages from the ear, preceded by dizziness and sharp pain behind ears and intense frontal headache.

Menstrual function deranged since last confinement, having been suppressed for three months, previously scanty and irregular. During August, 1898, and ten months following, the author, together with three medical brethren, had the privilege of witnessing several attacks, and had been unable, in the examination immediately following, to discover any abnormal condition of the tympanic membrane, and only a slight congestion of the cartilaginous portion of the external auditory canal. The hemorrhage was preceded by an exudate of a whitish fluid. Then followed slight clonic spasms of hand and arms, pupillary reflexes slow, pulse slow and small, but no loss of consciousness, attack lasting about five minutes. She had had as many as seven such attacks in one day. Patient thought attacks occurred during menstrual period.

A Case of Hemorrhage from External Auditory Canal. C. W. RICHARDSON (Washington, D. C.) *Annals of Ophthalmol. and Otol.*, 1896, Vol. V., No. 3.

The author reports a case of hemorrhage from a macroscopically healthy external auditory canal. Patient was a negress, age 30, a prostitute and first presented herself in May, 1893, with a sore throat. Examination revealed tertiary ulcers upon tonsils and pharyngeal walls, positively leucic in character. About a year after these lesions had healed, patient complained of bleeding from left auditory canal, attacks coming on nearly every week, lasting two to three days. This continued six months. During the seventh month hemorrhage became profuse, intermitting only four days. The month following and up to present date, bleeding had been continuous, with slight exacerbations and after careful observation to eliminate malingering, he averages the amount at eight grams daily, bearing no relation to the menstrual function.

The pinna appeared normal as did the external auditory canal. The membrana tympani had the appearance of a membrana in chronic catarrhal otitis media, having been intact since his first observation of the case. The canal had always been free, and the Eustachian tube patulous throughout the attack.

Pain was a marked subjective symptom. Spontaneous pain over temporal bone and pressure over this area or manipulation of auricle caused severe suffering.

Tinnitus had increased steadily with attacks of vertigo and progressive deafness (4-40) all limited to the affected ear. Pain was most severe just preceding hemorrhagic exacerbations.

The author points to the syphilitic history, suggesting that the blood exudes from the ceruminous glands, influenced by the improperly functioning cervical sympathetic.

Treatment included anti-syphilitic, electrical, local and general hemostatic with no improvement.

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DISCUSSION.

DR. J. G. DORSEY (Wichita, Kan.).—I was very much interested in Dr. Goldstein's report, for I had a case very similar last fall. A girl of 16 was brought to me for on-coming deafness in both ears. I

washed some epithelial scales from the ear with the syringe, and three days afterward spontaneous hemorrhage from the left ear occurred. The girl developed some hysterical symptoms. For two months this occurred, but I could not find the place whence it came. The parts all appeared to be normal. There was complete bilateral deafness. I will say that prior to the hemorrhage from the ear—in fact prior to my seeing her at all—she had hemorrhage from the stomach, but from that time there were no other symptoms of general hemorrhage. The case was under my care for about eight weeks. It was not accompanied by vicarious menstruation. She menstruated twice, as normally as usual while under my care, which seemed to have no effect on the hemorrhage.

DR. CONKEY.—While this paper was being read, a similar case was presented to my mind, yet different in many ways. I saw this case in consultation, and saw it several times. The history of the case was that there had been hemorrhages and watery discharges from the external ear. On examination I found an absolutely normal condition, both of the auditory canal and of the membrana tympani. The patient had had headache for several days. I referred the case back to the physician and told him as far as I could see nothing was wrong with the patient. The hearing was good. A few days afterward the doctor reported she had another hemorrhage, and from that time on he reported repeated hemorrhages. I did not see the case for about a month. I was called one night and found her in an apparently critical condition; suffering intense pain. I examined her again and found no lesion whatever and no inflammatory condition. I thought at first there might be a brain lesion. I examined the retina and found no trouble at all. I told the doctor I believed we had a case of hysteria, but I was in some doubt. For several months this woman continued to have these attacks, followed by discharges, and with the discharges there was relief. Neither I nor the attending physician ever saw the discharge. Finally she was sent to a hospital where I found her complaining of great pain in the ear and symptoms simulating paralysis. Upon pressure over the mastoid there seemed to be some tenderness, and although I believed the case was one of hysteria it seemed to me that strong measures were needed to get her out of the state in which she had fallen. It was a desperate case and required desperate measures. I advised a mastoid operation. I told her this must be done, and if necessary I would open the brain cavity. This statement had the desired effect upon her. From that time on she began to improve. She was soon able to leave the hos-

pital and since then (six months ago), I have not heard from the case. I believe it was hysterical.

I wish to add further that at the time of the last examination there was considerable dry blood in the external auditory canal, although there was no fluid.

DR. VAIL.—I have seen one case of hysterical hemorrhage from the ear, in which the patient was caught in the act of wounding the ear by means of a hat pin. Of course as soon as this was discovered, she was accused and put under surveillance.

The case of Dr. Goldstein's presents such peculiar and unique features that there is an uncertainty, in my mind at least, as to whether it was hysterical or not. I cannot understand how the wounding of the ear could produce such profuse discharge of sero-sanguinous fluid. The secretion was found to be something of the nature of cerebro-spinal fluid stained with some blood corpuscles. We are any of us liable to run across this condition, and I wish the doctor in closing would give us his opinion as to what he thinks it was.

DR. GOLDSTEIN.—I wish to ask Dr. Dorsey and Dr. Conkey whether provisions were made in their respective cases here reported for the definite exclusion of simulation, or the wounding of the external parts by the patient, or the introduction extraneously of blood or other fluid?

DR. DORSEY (answering Dr. Goldstein's question).—My case was in one of the catholic institutions, and I had a sister delegated to watch this girl carefully. It was more likely to appear when she was with people than when alone. The hemorrhage was abundant and more than could be produced by a slight injury of the ear. It was like a hemorrhage from the nose. I have seen the ear filled with blood. I immediately inspected it and found no place where blood could come from.

DR. CONKEY (answering Dr. Goldstein).—In this case there was no reason for malingering. She was at home and a poor woman. While I made no provision to prevent it, there was no real object in it.

DR. GOLDSTEIN (closing).—As far as I have studied this case, and I have tried to gather all details as completely as possible, one of the most potent and unusual features was the absolute exclusion of simulation and of the extraneous introduction of any fluid or substance.

Quite a number of cases have been reported of hemorrhage or exudate from the ear, with intact membrana tympana, where malingering was determined as the cause. In some of these cases of an

intensely hysterical type, the patient perhaps unconsciously provoked such hemorrhage either by injury to the auditory canal or by the introduction of fluids or irritants into the ear by way of the canal.

Another puzzling feature in my case was the determination of the character of the fluid. After careful and repeated examinations, it was decided that this fluid was not cerebro-spinal in character, because neither the specific gravity, the chemical reaction nor the microscopic appearance indicated it. The examination showed specific gravity 1.040 , the fluid of the consistency and character of serum, containing a few red blood corpuscles, and polynuclear cells.

Even with the accurate data at our command in this case, I have been unable to reach any definite conclusions or diagnosis. I might advance as a theory that of a neurosis of a vaso-motor type, where an intermittent paralysis localized in the mucosa of the tympanum would allow of a sufficient osmosis or transudation of serum through the capillary blood vessels until the middle ear cavity was filled to a sufficient extent to allow the fluid to syphon out through some mechanical or congenital crack or fissure into the external auditory canal. An intense hysterical temperament like that possessed by my patient would further predispose to some such vaso-motor irregularity.

I was careful not to advance any hypothesis or theory in reporting my case, because I thought it would detract from the actual value of the report. As a theory, the one which I have just stated occurs to me as the only one which may have a possible bearing on the etiology.

SOME CASES OF ASTHMA TREATED BY REMOVAL OF THE MIDDLE TURBINATE.

BY T. W. MOORE, M.D., HUNTINGTON, W. VA.

In looking over the subject of Asthma associated with intranasal disease I find that to Voltolini¹ is due the credit of arousing the interest of the profession to their relationship, but J. N. Mackenzie² has called attention to the fact that their association had been observed over two centuries before Voltolini reported his case; which was that of a man aged 33 whom he states was a constant sufferer with asthma and was cured by the removal of the polypi that filled both nares. As his book, in which the case is reported, was completed only a few months afterwards and the patient sent back to Ems in less than a fortnight after the operation, I think in the light of our present knowledge the permanency of the cure is not proven.

Hack³ reported 81 cases treated by intranasal operation on the inferior turbinates and septum, or removal of polypi. Twenty-five of these were not considered in his essay owing to the short time after the treatment. Thirty-three were cured and had remained so at the expiration of three years, seventeen were much improved and twelve not improved.

McBride,⁴ Kyle,⁵ Bosworth,⁶ and numerous other writers cite cases benefitted or cured by the removal of nasal polypi. Roe⁷ speaks of a case cured by the removal of hypertrophied tissue but does not state in what part of the nasal cavity this tissue was located, and any number of authors tell us of cases cured by removal of the intranasal disease, but no one, so far as I am aware mentions the diseased middle turbinate as an etiological factor or treatment confined to it as a remedial measure. This seems strange when we consider the great number of cases helped by the removal of polypi and remember that their most frequent attachment is the middle meatus and middle turbinate.⁸

Macdonald⁹ stands alone in claiming to have never seen a case of asthma benefitted by operation where the obstruction was in the middle meatus.

In my cases I was governed wholly by the diseased condition of the nose; to be more explicit, the intranasal condition demanded treatment disregarding the asthma, therefore my cases all come under the first of Schmiegelow's¹⁰ three rules, which freely translated reads,—“When the clinical picture leads to a belief that the abnormal condition of the nasal cavities is a factor in the production of

the asthmatic attack, which is to be inferred, when the asthmatic symptoms occur, or are aggravated with any increase in the nasal symptoms."

Case 1, female aged 16, was brought to my office in October, 1899, to be treated for nasal obstruction. I found both nares filled with polypi attached to the middle turbinates and middle meati. Patient complained of attacks of asthma which she had daily and had had more or less frequently since infancy. I removed the polypi as thoroughly as patient would permit, and repeated the operation three times during the year, the last two times I found them only in the right nostril. At the fourth operation I removed the anterior end of the right middle turbinate and forthwith the attacks of asthma became less frequent, of shorter duration and less severe.

Six months later the patient returned asking if I could not cut more out of her nose and relieve her asthma entirely. I then removed the remainder of the right middle turbinate with the result that the patient reports in February of this year two attacks of asthma in the two years since the last operation, both occurring in December, one in 1901 and one in 1902, while suffering from a severe acute coryza, these being the only attacks of cold she has had during this period. The patient's paternal grandmother and an aunt were sufferers with asthma.

This is the youngest patient whom I know has been practically cured by intranasal operation.

Case 2, Mrs. S., aged 36 years. One sister has hay fever and asthma. This patient was first afflicted when two and one-half years old and has suffered more or less ever since, attacks increasing in severity and frequency as she grows older until at the time of operation she had not had a single night entirely free from asthma for several years, often sitting for days in a chair, suffering almost continually.

Her family physician brought her to me July 31st, last year, stating that he had exhausted his resources and wished me to examine her nose and see if there was anything abnormal. I found the nares very large and both inferior and middle turbinates greatly hypertrophied and crowded against the septum. I removed the anterior halves of both middle turbinates, expecting to cauterize the inferior ones at some subsequent period. The patient had been suffering more than usual for several weeks. The operation relieved her completely and for six weeks she was entirely rid of the dreaded attacks, then a recurrence began, the paroxysms increasing in frequency and severity until November 11th, when I removed the re-

mainder of the middle turbinates, with the result that patient was again completely relieved until in December when they returned with their old severity and in February they were quite as frequent as ever.

Unfortunately I was unable to control the pain with cocaine during the second operation and patient refused to allow me to touch the inferior turbinates badly as they needed treatment.

Case 3, Mrs. C., aged 32, no history of asthma in family. Patient suffered almost constantly from asthma from March, 1900, until operated upon in May, 1901. She was first afflicted while suffering with influenza accompanied by the usual severe rhinitis. This occurred about the seventh month of pregnancy. The attacks abated during her confinement but came on two days after labor and continued with greater or less severity.

Her physician at several times despaired of her life. After the operation she was sent to the country where she remained nearly three months improving all the time and returning home entirely free from asthma until the following April, when she contracted a severe cold and again suffered with her old affliction for ten days. Since this she has not been troubled excepting brief paroxysms occasionally which last only a few minutes and are relieved by sneezing which is usually accompanied by slight bleeding from the nostril operated upon.

The operation in this case consisted in the removal of the right middle turbinate. This was followed by a few drops of pus in the hiatus semi-lunaris, probably from the frontal sinus.

Case 4, Mrs. I. E. C., aged 27. Mother is asthmatic. Had attacks two or three times weekly never being spared more than three weeks, had suffered for nearly four years prior to the operation of removal of anterior ends of both middle turbinates in August, 1900. The improvement began at once and until the present she has not had attacks oftener than one in two or three months, her last one occurring in December. They are milder and of shorter duration.

I have operated upon six other cases where the patients had asthma three or four times yearly. Two are cured after two years. Two cases are improved; one has not reported and sufficient time has not elapsed since the last one was treated. In three of these the inferior turbinates were either cauterized or a portion excised.

Now to what conclusions do these cases lead us as to the relationship of asthma and intranasal disease. I trust that I may be pardoned if I quote from an editorial written in 1890¹¹ for it so aptly and clearly expresses my views.

"All that may be said is that wherever the nasal branches of the trigeminus spread out in the nasal cavities, there will be found spots, irritation of which produces a reflex and in certain cases where resistance of the medullary ganglion is lowered, or their excitability increased, there results, instead of an ordinary simple reflex of the respiratory center such as sneezing, an overflow of nervous force, which passes down the phrenics to the diaphragm and possibly overflows to the vasomotor nervous cells in relation with these parts. What is very certain and admitted clinically is that there must be some abnormal state of excitability of the basal ganglia, which is vaguely understood under the terms neurasthenic or neurotic."

In my cases the only after-treatment was some bland oil, usually sabalol, as a spray, excepting two cases in which I gave an old asthmatic preparation containing

R	Tr. hyosciami	
	Spt. æth. comp. aa	30.00
	Potassii iodidi	15.00
	Syrupi	30.00
	Aquæ menth. pip. ad	150.00
Misce.	Sig. Two teaspoonfuls in water every two or three hours.	

This I have found one of the best antispasmodics in such cases.

I believe that the benefit derived by these patients was due to allaying the irritation to the nerve endings in the nose and very little if any to suggestion.

Case 2 had developed an instability of the control center and whilst the operation gave relief for awhile the debilitated condition of the nervous system needed only an irritant to again bring on the spasms, this was supplied by an attack of influenza.

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¹¹ *Journal Laryngology*, 1890, pages 101-106.

DISCUSSION.

DR. J. W. MURPHY (Cincinnati).—This subject of asthma has given us all a good deal of worry. No operation seems to be a panacea for the cure of the disease. The exact cause and etiology of this disease seems as obscure to-day as ever it has been. I have operated on quite a number of these cases for this condition and now and then a case seems to be materially benefitted, but in the majority of cases the condition returns. There seems to be a vaso-motor cause here and most any operation seems to benefit the case for a short time. I have at the present time a gentleman who is a great sufferer from asthma and I have operated on him three or four times. Every operation helps for a while; but there is nothing more to operate on and I do not know what we will do when he comes back again. I think it is Bosworth who claims that 80 or 90 per cent of asthmatic cases can be benefitted by operation, but my experience has not been such. I think in a great many of these cases there must be a great deal in suggestion. I had a case under observation several years ago in which a large spur was removed from the septum and the nose was put in as good condition as I was able to, but the attack of asthma returned in a few months as severely as before. Then the patient went south and the climatic change helped the condition, but as soon as the patient returned from Texas the asthma returned.

DR. VAIL.—Dr. Murphy's remarks almost explain themselves. He said that a great many of these cases are amenable to suggestive therapeutics. The suggestive treatment is the very thing they get from the Osteopath. The Osteopath will generally claim that the "spheno-palatine" duct is closed, or that the "25th cervical vertebra" is out of joint, and so impress the patient with these wonderful things.

I knew a young medical man who had suffered for years with asthma. About three years ago he took treatment from an Osteopath who informed him that his ensiform cartilage was out of joint. One treatment cured him—so he stated to me. He was so impressed with the tenets of the osteopathic school that he turned osteopath himself. Lately I have learned that his asthma is as bad as ever. I presume that familiarity with the workings of osteopathy has staggered his faith to the extent that he is no longer a fit subject for their suggestive treatment.

DR. GOLDSTEIN.—There is no doubt of the fact that hypertrophied turbinals occasionally play an important part in the etiology of localized asthma, and that occasionally operative interference will give you the radical result you desire. To me the most important feature

would be to determine just when to operate and just when to let this condition alone.

DR. W. R. MURRAY (Minneapolis, Minn.).—I believe a number of these cases can be classed as neuroses, but a large number depend on nasal lesions and the removal of these will often result in a cure. I am not prepared with statistics, but I have operated on a number in the past four years, and I endorse the writer's views as to the middle turbinal being frequently the seat of the disease. I cannot say how permanent the relief is after operation, but do not recollect any case that has come back where the asthma was relieved at the time. But sometimes we do not get a cure of the asthma after removal of the lesions of the nose. I remember one case, the most violent I had ever seen, where the man had been subject to attacks for fifteen years. He had a prominent nasal lesion. The middle turbinals were hypertrophied and there were nasal polypi, also enlarged inferior turbinals. I operated, but the asthmatic attacks continued and he has suffered practically as much as before the operation. I remember six or eight cases in the past three or four years where the attacks have disappeared within a week of the time of the operation, and as far as I know, in those cases none have recurred.

DR. BARNHILL (Indianapolis, Ind.).—We in Indianapolis live in the center of the rag-weed district of the United States, as well as in the center of its population, and therefore any paper on the subject Dr. Moore has just presented, becomes of the greatest interest, because here in Indiana the rhinologist sees annually a large number of cases of asthma and hay asthma.

Some years ago I read a paper on the same subject, and being aware of the criticism that had been made of the surgical means of treating asthma, I attempted to ascertain if possible, if there was any easier, pleasanter or more certain means of cure. I therefore wrote to each professor of general medicine in each leading regular medical college in the Middle west, asking if he had ever had a case in which he had cured a case of hay asthma by drugs alone. In every answer received the report was negative. Such is not the case with surgical means, for many rhinologists have reported cures following well directed intra-nasal surgery, coupled with careful general medication. I believe the region of the middle turbinate to be most often at fault in these cases, and that by removing areas of pressure, and securing better drainage from the accessory cavities which open under the middle turbinate body, we can secure good and often brilliant results. Much has been written about the nervous element which accompanies these cases. I believe this is often a result of the nasal pressure and faulty sinus drainage, for certainly judging from the amount of local

suffering endured by these patients it would not seem unreasonable if their nervousness was attributed as a result of the nasal condition, and not a causative factor of the asthma. I have recently removed the middle turbinate, together with a large number of concealed polypi in such a case. Several years ago I had removed several polypi from the same case, which were large enough to be seen in the nasal fossa. This helped, but the sneezing, and asthma continued unabated. Several times since polypi were again removed with like result. Recently I insisted that the patient permit more radical methods, which was done, and I removed the middle turbinal, which exposed many polypi springing from the ethmoid and about the ostium maxillaire. These were also removed, and the result is entirely satisfactory to the patient, and I believe a cure will result. Certainly in cases where there is a clear condition of nasal obstruction and faulty sinus drainage, our best results must come through surgical methods.

DR. PYNCHON.—I personally am disposed to think that asthma is chiefly due to the traveling downwards of secretions from the post-nasal space. Why such secretions do not always produce it, but do in certain cases, I cannot say. Enlargement of the middle turbinal is acknowledged by all to be a factor in hay fever. In my paper yesterday I stated that enlargement of the middle turbinal impairs the ventilation and drainage of the attic of the nose. By this impairment is produced catarrhal sinusitis, the secretion from which travels to the post nasal space, as emphasized by Cobb. Dr. Moore stated that he did not know of another writer who had called attention to middle turbinal hypertrophy as a cause of asthma. In a paper I read at New Orleans four years ago I called attention to this fact, and emphasized it in the paper I read at Cincinnati two years ago.

DR. GOLDSTEIN.—Last July or August Dr. G. B. Holt of New York, one of the older surgeons of the Manhattan Eye and Ear Hospital, published a paper in *THE LARYNGOSCOPE* on favorable results in the removal of turbinal hypertrophies in the treatment of hay fever.

DR. MOORE (concluding).—I am very glad to have the indorsement of the surgical treatment of this disease by so many distinguished members of the Academy. I think that unless we watch these cases closely afterwards we are apt to think that they are cured when they are not. When I started this paper I was sure that three of these cases were cured and I did not know until I interviewed the patients that they had had any subsequent attacks.

I think that the middle turbinate is an important etiological factor, but we may remove the cause as we often see in traumatic epilepsy and still the neurosis will remain.

A SYNOPSIS OF MY FIRST HUNDRED MASTOID OPERATIONS.

BY C. BARCK, M.D., ST. LOUIS.

Although larger statistics have been published, it is at times beneficial to review the subject from the standpoint of personal experiences. These cases form a continual series, operated within the fourteen years from March 1887, to January 1901. Most of them were patients in private practice, and the majority were operated in residences, the others in hospitals.

STATISTICS.

Of the 100 cases, 91 recovered and 9 ended fatally.

There was a slight preponderance of the left side over the right, the left being affected 51 and the right 49 times.

The ages varied from 4 months to 63 years; 42 were below the age of 15, among them 5 babies.

Two cases were "primary" mastoiditis, without previous involvement of the middle ear.

Bezold's form was encountered twice.

Cholesteatoma was found in 5 instances.

Caries with exfoliation of the entire labyrinth occurred once.

For osteo-sclerosis one operation was performed.

Subperiosteal abscess was present at the time of the operation, 14 times.

Facial paralysis was met with in 5 cases.

The healing process required 4 weeks in the shortest instance, and nearly 6 months in the longest.

Subsequent operations became necessary in 6 cases; a second in 4, and a second and third one in 2.

Erysipelas followed the operation twice, both cases ending in recovery.

The intracranial complications were as follows:

Subdural abscess, 2.

Sinus thrombosis, 5.

Brain abscess, 3.

Meningitis, 4; among them 2 were tubercular.

In one of the above cases there was sinus thrombosis and cerebellar abscess present.

Of the nine cases that ended fatally, none died from the effects of the operation; all of them were cases with intracranial complication. The latter were: Sinus thrombosis twice, once complicated with a deep cervical abscess. Osteophlebitic pyemia without sinus thrombosis, once. Abscess of the brain, twice. Meningitis, four times.

A number of these cases were seen in the eighties, before the era of intracranial surgery for ear complications, which has developed since 1890, and therefore no attempt was made to go beyond the mastoid proper. In five of the nine cases a post-mortem was held by me. At the end of the article will be given a report of the cases with intracranial complications, both the fatal ones and those which recovered.

The affections of the babies (below one year of age) were all acute cases, most of them robust children otherwise. All made a quick recovery, and the after-treatment was less unpleasant than expected.

In the two cases designated as "primary mastoiditis" the closest examination failed to reveal the presence of an otitis media. In both, the abscess was located in the outer mastoid cells; the antrum was opened, but found intact. These cases might be classed as osteomyelitis of the temporal bone.

The percentage of Bezold's form seems to correspond to the experience of others. That the drainage in these cases is difficult, is well known.

The five cholesteatomata form a small percentage, whilst they form a considerable one in European clinics.

The case of caries with exfoliation of the entire labyrinth was reported in full at the meeting of the American Medical Association at Milwaukee, in 1893.

A typical case of "osteosclerosis" was met with once. A lawyer from Kansas had had an acute suppuration of the middle ear several months before he came to St. Louis. According to his statement, the mastoid region was at one time tender on pressure and somewhat swollen during the height of the otitis. Although the latter healed under treatment by his physician, a severe pain in the mastoid region had persisted. This was constant, robbed him of sleep, and caused in time a severe melancholic depression of mind. On his arrival, I found the tympanic membrane normal, with the exception of a cicatrix. Hearing nearly normal. No swelling, no redness, no pain on pressure upon mastoid; only the mentioned severe neuralgia. The diagnosis "osteosclerosis" was confirmed by the operation. The entire mastoid was eburnised,

with a minute antrum left. The operation gave immediate and permanent relief. This coincides with the experiences of others.

Of the five facial paralyses, two were present when seen for the first time. One of these recovered entirely within one year; the other one improved slowly. The patient was a boy of four years when operated in 1889, and now at the age of eighteen shows hardly a trace. The third occurred in the case of caries just mentioned, during the period of exfoliation of the sequestrum. The paralysis was quite a complete one for some time; gradually the upper branch, controlling the orbicularis muscle, regained its power, so that the eye can now be closed nearly as well as the other. The lower branch, however, shows a considerable paresis still. The fourth and fifth cases were due to operative procedure. One was a mild paresis, after the evacuation of a subdural abscess. Contrary to expectations, it receded very slowly, and now, after the lapse of over two years, the last remnants show themselves at the angle of the mouth, during the act of laughing. The other occurred during the second operation for a large cholesteatoma with cerebral symptoms, performed after the radical method. The palsy was total for some weeks; then the upper branch recovered slowly, so that the eye can be closed now with an effort, while the lower part of the face of that side is still, after six years, nearly entirely paralyzed. I have dwelt at length on these cases and their course, because all of them have been seen at intervals up to now, a rare occurrence, as they are usually lost sight of.

I have not included among the foregoing, an instance of facial paralysis, where an erroneous diagnosis was made as to its cause. There was present a chronic otorrhea and facial paresis, but no tenderness or swelling over the mastoid. It was supposed that a carious process in the neighborhood of the Fallopian canal caused the affection of the nerve; the mastoid was opened but found intact. The patient died about half a year afterwards and the post-mortem revealed sarcoma of the brain. The paralysis had been a central one.

By "subsequent" operations I do not understand the repeated scraping out, removal of sequestra, etc., before the closure of the primary wound, but instances, where the patients had been dismissed and returned years later with a mastoid affection of the same side. Apropos to this I would like to report a rather remarkable history.

A. S.—The girl was first seen, when 9 months old, on March, 20, 1892. After an otitis media of one week's standing, a typical mastoiditis had developed. As it did not recede under the usual methods, I operated on April 3, 1892. Small amount of pus and

detritus, mainly in outer mastoid cells. The healing was normal, the wound was closed on May 23, 1892. The discharge from the ear had ceased prior to that. The child was in perfect health during the next three years.

In March, 1895, an acute otitis developed in the same ear. In spite of an immediate paracentesis, the mastoid became involved again very rapidly, and was opened the second time on March 28, 1895. Again normal healing. Suppuration from the middle ear ceased about May 15; mastoid wound was closed on June 2, 1895. Hearing, just as good as on the other side. The child was robust and well developed. It was seen at intervals and the ear as well as the mastoid remained in good condition for the next 4 years.

In April, 1899, the course of 1895 repeated itself. Earache for just one night. When I was called the next morning, I found the symptoms of an otitis media and the old scar on the mastoid already somewhat red. In spite of an immediate paracentesis, the mastoid symptoms developed so rapidly, that I had to open it for the third time on April 9, 1899. A small amount of fluid pus was found, besides granulations and cheesy masses. The cavity was cleared with the utmost care and every focus of softened bone was curetted. The detritus removed was sent to a bacteriologist to be examined for tubercle bacilli. He reported a negative finding. The wound was again closed June 22, 1899, the otorrhea having ceased. Hearing about $\frac{2}{3}$.

In May, 1900, the family desired to make a trip to Europe, and brought the child to me a week before their departure. It was in very good general health, mastoid perfectly sound; drum membrane of normal appearance, hearing about as good as in the other. Therefore, I saw no reason why they could not take the child along. After being six days on the ocean, the same course as in '95 and '99 took place. When they arrived at Bremen, they consulted a physician at once, who advised them to proceed to Berlin. There the child was operated immediately by one of the foremost specialists, but died 3 days later. According to the history given by the father, infective lateral sinus thrombosis was the cause of the fatal end.

There is still doubt in my mind, whether or not this case was of a tuberculous nature. The negative findings of the carious masses mentioned, does not, of course, exclude it. At one time, during '99, there was some cough and the family physician diagnosed an infiltration of the apex of the right lung. The examination of the sputum, however, also proved negative, and the cough ceased entirely after several weeks. There is no tuberculosis in the family

of the father and a very distant one in that of the mother. The other two children of the family are perfectly healthy.

In the two cases, where erysipelas followed the operation, the latter had to be performed in a hurry, in private houses, and with unclean surroundings.

The histories of the 14 cases of intracranial complication are as follows:

The two patients with "subdural abscess" recovered. In one of them, an intracranial affection was suspected, in the other it was found unexpectedly.

Case 14.—M. R., æt. 63. Very violent otitis media but with spontaneous perforation. When seen several weeks later, he presented the typical symptoms of an acute mastoid abscess and was in a comatose condition. The hearing in the affected ear apparently entirely gone. No changes in fundus of either eye. Operation next day. External shell of bone 4 mm. thick and very dense. Antrum filled with pus. Tegmen antri greenish, discolored and partially destroyed. Above it, an abscess cavity, $\frac{1}{2}$ cm. in diameter. Clearing; regular recovery.

Case 93.—A. O., æt. 35. Chronic otorrhea of about 15 years standing. Middle ear filled with granulations, and chronic mastoid affection, but no cerebral symptoms. The operation revealed large destruction of the bone, close to the lateral sinus; and the tegmen antri wanting to a large extent, especially in the direction of the tegmen tympani. A subdural cavity contained nearly a teaspoonful of exceedingly fetid pus, which caused suspicion of a cerebral abscess, but careful probing could not detect a perforation of the dura mater. During the healing process, a portion of the bone around the Fallopian canal became necrotic, and its extraction was followed by a mild facial paresis, with a very slow recovery, as stated above.

Of the five cases classed under the head of sinus thrombosis, one which was not operated upon, ended fatally. This was rather an instance of otitic pyemia as there was no thrombus found in the sinus at the post-mortem. (Case 43.) The other four were operated upon, and two recovered. Of the two which succumbed, one was operated at too late a stage, and the other one died really from an unrecognized deep cervical abscess.

Case 43.—J. R., æt. 23. Influenza and pneumonia for three weeks. The right ear had been running for a short time before the influenza set in (possibly the first localization of the disease). During its course the left ear began to discharge also. As there had never been any pain in the right ear, there was no particular atten-

tion paid to it until mastoid symptoms had developed. When seen in consultation with the family physician, the mastoid region was red and edematous. The upper posterior wall of the external canal was sagging to such an extent that the swelling closed its lumen nearly entirely. Severe pain in mastoid region and the entire head.

Operation next morning. Bone sclerosed; outer shell exceedingly hard and dense. No cavities until the antrum was reached. In this fluid pus was encountered and a communication was found connecting with the abscess cavity projecting into the external auditory canal. Free communication with tympanum.

For the first three days, the course was a good one. Free from pain. Temperature fell below 100° F. Was sitting up on the second day.

On the fourth day he had a chill. Temperature rose to 104° F. Wound carefully inspected without unusual findings. Temperature fell and was normal after 36 hours. It remained so for the next 5 days, then another sudden chill took place, the temperature rose to 104 $\frac{2}{3}$ ° F. in two hours, accompanied by profuse sweating. During the next few days, the temperature did not sink below 102° F., frequent chills and vomiting. For the last two days severe pain and some swelling in left shoulder joint and elbow joint. Patient grew weaker and weaker and died two weeks after operation.

Autopsy—Entire temporal bone removed. No thrombus in lateral sinus. The blood in it was fluid, but mixed with some minute particles of pus. The bone separating it from the wound was in its thinnest part 2 mm. thick, dense, and healthy looking. The entire petro-mastoid bone was of hard, ivory like consistency, but traversed by a number of wide veins filled with fluid pus. Contents of the cranial cavity normal. In this instance, an opening of the sinus could hardly have saved the patient.

The next two cases have been published in my article, "Two Cases of Opening of the Lateral Sinus for Infective Thrombosis," with a table of operations performed previous to 1897. (*Annals of Otol.* etc., 1897.)

Case 45.—J. K., æt. 26. Hungarian by birth. Patient had since childhood, left-sided otorrhea, the cause of which was unknown to him. No treatment had been resorted to, as he experienced no pain. Three years ago he was seized with a very violent pain in the ear, for which he was treated by a specialist for about four weeks. Since then, a somewhat fetid discharge has persisted, but no pain until recently.

Patient first consulted me on January 15, 1894, because of pain in the ear for three or four days preceding. He is of medium size and

fairly well nourished. Right ear normal. Left ear, foul smelling discharge in moderate quantity. After cleansing, the walls of the external canal were found to be somewhat swollen, the tympanic membrane gone, and the middle ear completely filled with small polypi and granulation tissue. The region over the mastoid showed no swelling, but was somewhat tender on pressure. Temperature 100° F.

In consequence of careful cleansing with antiseptics for about a week the pain subsided, and the temperature fell to normal.

I then removed two of the largest polypi with the snare; but finding the entire middle ear filled with granulations, proposed an evacuation of the cavities by an operation through the mastoid. As the patient felt very well at the time, he declined, and I saw him only once or twice within the next two weeks. On February 17, I was called to see him at his residence. He was confined to bed, complaining of severe pain in the entire left side of the head. He had had chills and rigors for two days, and had vomited two or three times. Temperature 100°, pulse 100. The region over the mastoid was somewhat swollen, and the tenderness on pressure was more pronounced. I informed him that an operation was urgent, and sent him to the hospital the same afternoon.

Operation (Stacke's), February 18th.—Immediately after the removal of the external table, fetid pus was found. The antrum was exposed, and found filled with granulation tissue and pus. A portion of the posterior wall of the external canal was then removed, so that the canal, antrum and tympanum formed one large cavity, from which the foul smelling pus and abundant granulation tissue were carefully removed. Only small portions of the ossicles remained. The tegmen tympani and antri were found healthy, but the bone in the region of the lateral sinus seemed somewhat discolored, so that I stated at the close of the operation that, if the symptoms did not subside rapidly, opening of the sinus would be indicated.

After the operation the patient felt relatively well and the temperature did not reach 100°. However, during the next two days it rose to between 103° and 105°, the pulse varying between 80 and 112.

He complained of headache and had a chill. There was no paresis nor any material change in the fundus of the eyes. The general condition and curve of temperature were typical for systemic pyemic infection. Therefore,

Opening of the lateral sinus, February 21, in its sigmoid portion, from the cavity in the mastoid. An oblong piece of bone, (discol-

ored as before described) nearly one inch long and one-third of an inch wide was removed by careful chiselling and forceps. Some pus and granulations were found between it and the exposed wall of the sinus, which was also discolored. The sinus was then punctured with an exploring needle and found thrombosed. An incision in its wall, as long as the opening in the bone, was then made, and a large softened purulent thrombus removed. The cavity was then gently cleansed with a blunt spoon as far as practicable, first downward, then upward. There was no hemorrhage from the lower, and only a moderate one from the upper end. As soon as this appeared the sinus was closed by compressing its walls with antiseptic gauze and the hemorrhage easily checked.

Subsequent course—The expectation of checking the further infection of the system was not realized. While on the next day he had no chills (temperature below 100°), and felt some better, on the following day he became worse, the symptoms beginning with a chill. The temperature again rose to $104 \frac{2}{5}$, pulse became frequent, pain appeared in different joints, and, in short, he presented for the next two days the typical picture of an even more serious pyemic infection.

The area over the jugular vein on the neck had been regularly examined since his entrance into the hospital, but it was never painful, and no thickening nor stringiness indicating thrombosis of the jugular vein could ever be detected. For the continuance of the septic state there seemed to be but one explanation, viz., that the septic thrombus had already reached the bulb of the jugular, and had not been entirely removed. It was accordingly decided to ligate the jugular vein, an operation which was done by Dr. A. C. Bernays, on the 24th. The vein was filled with normal blood; no sign of thrombosis. It was ligated in two places and cut between the ligatures.

The ligation did not have the desired effect, as the pyemic state continued. Frequent and profuse perspirations, pain in the joints and high temperature weakened the patient more and more. He became delirious, then somnolent, and finally fell into deep coma, and exitus lethalis ensued on the 15th of March. The temperature during the last two weeks ranged between 101° and 105° , pulse between 110 and 130.

The wound in the mastoid and sinus was throughout in the normal condition of an undisturbed healing process.

Post Mortem—The skull was opened in the usual way. The contents did not present any pathological appearance, and the brain was intact. There was no lepto-meningitis, nor any trace of pachy-

meningitis. The dura mater covering the temporal bone was perfectly normal throughout. The inner wall of the lateral sinus in its sigmoid portion was of normal appearance, half transparent, so that the defect in the bone due to the operation could be plainly seen through it. Upward, in its horizontal portion, the sinus was closed for about one inch, its walls being *soldered*, as it were. Downward, in the end of the sigmoid portion, it was patent for about half an inch, but further down it was firmly soldered. The jugular vein between the bulbous and the ligature was filled with a healthy thrombus which showed no disintegration, and which was without doubt the consequence of the ligature. No thrombus was present in any other of the dural sinuses. Thus far, the post-mortem findings did not offer any explanation for the continuance of the pyemic state after the two operations, and the conclusion arrived at at this time was, that the infection had become too general to be overcome by obliteration of its foci. And it was rather accidentally, than otherwise, that the true cause was discovered. Owing to the pressure of time I did not at first intend to remove the temporal bone in order to preserve the specimen, but afterward decided on doing so, and during this procedure, after the big muscular layers on the posterior portion of the neck had been divided, we found a large abscess under the deep fascia of the neck below the splenius capitis and levator scapulæ in the posterior cervical triangle. The abscess contained about two tablespoonfuls of exceedingly fetid pus. The digastric fossa was free from pus.

About an inch and a half upward, direct communication could be traced between the abscess and the lateral sinus through a very large mastoid foramen. This abscess was without doubt the cause of the continued pyemia and fatal termination. No symptoms pointed to its existence during life. No redness, no swelling, no pain in the region. There is no doubt that its recognition and surgical treatment would have saved the life of the patient. Not as an excuse, but simply as a matter of fact, I will state that the patient had been seen daily, not only by myself, but by the aforementioned surgeon, and also by one of the most careful diagnosticians in our city. In spite of repeated consultations, the real focus of the pyemic infection was not discovered until the post-mortem examination and even then was almost overlooked.

Case 52.—Geo. G., aged 26. Fell from a wagon, striking upon his head, on May 3, 1895. He at once became unconscious and was removed to the city hospital, where he lay in this condition for three days. His right foot, both hips, and the right side were bruised. A superficial scalp wound running longitudinally for about an inch, was

found, one and one-half inches above the right auricle. There was considerable hemorrhage from the right ear, which continued for a week and a half. The wounds were dressed and healed. The ear was cleansed with hydrogen peroxide and the patient dismissed from the city hospital on May 24.

When he had arrived home, he complained of dizziness, and acted and spoke in a manner indicating to his relatives that he was anything but well.

I first saw the patient on the following day, and found the condition present as follows: Patient felt dizzy, his sensorium was benumbed, answered slowly, and sometimes not at all. No difficulty in articulation. No paresis nor paralysis. Had had a rigor and chill the day before, and one that day. Temperature 102° , pulse 106. There was a fetid discharge from the right ear. The walls of the external canal were so swollen, especially the posterior and upper, that no part of the tympanic membrane could be seen. The auricle projected outward and there was considerable painful edematous swelling over the mastoid region.

Diagnosis:—Fracture through temporal bone and tympanum, otitis media, and secondary infection of fracture-line from the otitis.

Operation May 26th.—The fracture-line in the squamous portion of the temporal bone was easily visible after detachment of the periosteum, passing from behind forward and downward. Directly below the outer lamella of bone, and in the line of the fracture, there was an abscess as large as a bean. The antrum was found free. After cleansing of the cavities, some pus was noticed oozing slowly through a fine opening in the posterior wall of the cavity, which separated this from the lateral sinus. The bone was removed, a piece about three-fourths of an inch long and one-third of an inch wide. The wall of the sinus presented an irregularly oblong opening. I am unable to decide whether this was due to a violation during chiselling, or to the fracture. No hemorrhage ensued, as the sinus was filled with a thrombus. The opening in the wall of the sinus was enlarged, and the thrombus, which was softened and covered with pus, was removed. A small hemorrhage resulted. After a careful cleansing upward and downward with a blunt spoon, the walls of the sinus were compressed with gauze.

May 27. Patient had had no chill, was more rational, but still somewhat drowsy. Temperature below 100° since operation. Pulse never over 88.

June 1. Temperature rose, for the only time during after-treatment, to $103 \frac{2}{5}^{\circ}$, but fell rapidly after change of dressing. The recovery was otherwise an uninterrupted one. The patient became

perfectly rational four days after the operation. The wound was closed and the patient dismissed on June 26.

The discharge from the ear had ceased, and the perforation in the tympanic membrane had closed prior to that date. Hearing one-half at dismissal.

Patient has been recently heard from, and is and has been perfectly well since the operation.

Epicrisis.—Case 45 has been reported so minutely, because it is very instructive, and little needs to be added. Whether or not the destructive process had invaded the lateral sinus at the time I proposed the evacuation of the mastoid cavities, and whether or not the fatal termination could have been prevented by an operation at that date, is of course an open question. It would have been more advisable to open the sinus at once after the mastoid operation, and I shall certainly proceed on that line in future cases.

The other case is interesting on account of its course. The fall caused a fracture of the temporal bone, extending near or possibly into the lateral sinus, and also ruptured the tympanic membrane. The open tympanum was not protected sufficiently, and infective suppuration set in. From this focus the infection spread along the fracture-line, causing there at one point a small abscess, finally reaching the sinus.

Case 78.—S. S., æt. 37. Since childhood, otorrhea in right ear. Seen first in November, 1898. Profuse fetid discharge. Middle ear filled with granulations. Severe headaches and dizziness for two weeks. Staggered slightly and was not able to walk straight. Mastoid region slightly swollen and tender on pressure. Advised an immediate operation, but patient did not return.

On February 22, 1899, was called to the hospital to see patient. He had been treated in the meantime at a dispensary, and had been able to work from time to time. For the last two weeks, he had been confined to his bed.

Patient was very weak and emaciated, and presented the typical picture of pyemia. Temperature over 104° F. Profuse sweating. Severe headaches in occiput. Cough. (House physician found consolidation of a part of the left lower lobe of the lungs.)

Operation next day. After cleansing the mastoid and middle ear (Stacke's operation), the sinus was laid bare by an oval opening, about one cm. in length. The bone covering the sinus was discolored and disintegrated. The sinus seemed to pulsate at times. Of two explorative punctures, the first was negative while the second drew pus. The opening in the bony wall was then enlarged by the forceps to 35 mm. and a corresponding incision was made into the

membranous wall. The sinus was filled with a fetid, decomposed clot. It was cleared upwards until free hemorrhage ensued; then downwards into the bulb of the jugular vein. Moderate hemorrhage from this side. As there had been no indication of involvement of the jugular vein, it was not ligated.

The progress was a favorable one for the following three weeks. He had no chills any longer, and not much pain. The temperature fell to normal. The secondary abscesses commenced to develop on cheek and neck, one after the other. At the same time, there was a severe pain on the vertex of the skull in an antero-posterior line, spontaneous as well as on the slightest touch. The diagnosis of involvement of the longitudinal sinus was made. Chills became more and more frequent, temperature remained a constantly high one; cough increased considerably and finally a comatose state developed which ended fatally 5 weeks after the operation. No post-mortem permitted.

Case 94.—L. A., plasterer, æt. 55. Was seen on Oct. 17, 1900. He gave the following history: Otitis in May. Pain in ear, then running. Went to a dispensary for four weeks, in August. Always pain in that side of the head, increasing regularly; for last two weeks so severe that he could not sleep.

October 17, 1900. No discharge from the ear. No perforation of tympanum, but considerable thickening. Watch i. c., not much better in right ear. Mastoid region swollen and edematous, auricles standing out; severe pain on pressure. Advised operation at once; patient did not consent; ordered ice, etc. Patient called two days later but missed me and was not seen for the next two weeks.

October 30, 1900. Excruciating pains in the head, chiefly at night, so that he had hardly slept since; considerable remission during day time; did even work two days ago. Headache was felt in whole head, but referred mainly to the occiput. Touch and pressure everywhere in back of head exceedingly painful. Condition of ear and mastoid same as when seen at first. Temperature below 100. Stated that he had never had a chill, that he sometimes felt dizzy, but not at this time. Nothing abnormal was noticed in his gait, although a special examination as to this point was not made, as no brain abscess was suspected.

October 31, 1900. Operation under narcosis. Immediately after the incision there was noticed, at its upper and posterior end, a small fistula in the bone. The exploration of this was postponed until later in the operation. First, the mastoid was opened. Pus immediately below outer shell. Very large cavity filled with pus and detritus, especially downwards to the very tip of the mastoid, whose

mesial wall was somewhat carious. Also pus in the antrum. Sinus wall examined carefully; apparently normal.

The above mentioned fistula in the bone was $1\frac{1}{2}$ inches backwards and upwards from the mastoid opening, at the place where the three sutures meet. A small amount of pus was oozing out of it. Fistula was enlarged to a roundish opening, 1 cm. in diameter. All around it the dura and pia mater were adhering to the bone. They also showed a small perforation, through which the probe entered mesially into the cerebellum, 2 cm. deep. Dura incised and cerebellar abscess opened with knife. One and one-half drachms of yellow, absolutely odorless pus evacuated. On account of location of abscess, and the mentioned appearance of sinus wall from the mastoid wound, it was supposed, that in this instance the path of infection was not the usual one, viz., through the sinus, and therefore, the sinus was not opened. Abscess drained in usual manner.

Patient was free from headaches about one week. Temperature reached 100° F. only once; discharge daily about one drachm, decreasing steadily. On November 10, headaches again appeared, increasing in severity. These were first attributed to insufficient drainage. The drainage tube was, therefore, removed, the external opening of the fistula freed from the granulations which had sprung up, and iodoform gauze substituted. In spite of this, the headaches persisted and the temperature rose to 101° F. On November 29, some swelling and tenderness in neck was observed, and on pressure in this region, some pus escaped through the fistula, which proved connection. Careful probing discovered another fistulous path, mesially and downwards.

November 30. Second operation. First incision elongated downwards. Entered between the sterno-cleido and the splenius through the deep cervical fascia, till the finger could explore the region behind the mastoid tip. There was no pus found between the muscles. In the depths of the wound, the finger felt a soft membrane. On pressure on this, pus escaped through the fistula. It was evidently the bare inferior wall of the sinus, whose bony covering had been destroyed. The knee of the sinus was then laid free for $1\frac{1}{2}$ inches, by chiselling away the bone between the mastoid wound and the fistulous opening. Some pus around the sinus. The latter was incised in its entire length. It contained a small thrombus, which was removed and the sinus cleaned. There was a very small hemorrhage; apparently the sinus had collapsed and the walls were adhering, upwardly as well as downwardly. From the upper end of the opened sinus, the fistula leading into the cerebellum was plainly visible.

Subsequently, the recovery was an uninterrupted one. The discharge from the cerebellar abscess ceased in one week. The large wound healed by granulation, and was finally closed by a plastic skin flap, on January 22, 1901. The patient could be dismissed on February 17.

This case supports the standpoint taken lately by other authors, that, in searching for an otitic cerebellar abscess, it is always best to go through the sinus. The infection may travel through the labyrinth and from there by way of the internal auditory canal, or one of the aqueducts, but in more than 75 per cent it goes through the sinus. Although this point was taken into consideration during the first operation, as mentioned above, the conditions found, viz., the apparently healthy wall of the sinus, and the unusually high location of the fistula, spoke against it. It became evident during the second operation that the fistula led into the sinus, and through it into the cerebellar abscess, but since the dura was adherent all around the fistula, it was impossible to recognize the true condition.

As found so often, the symptoms of the cerebellar abscess were absolutely masked, the headache being sufficiently explained by the mastoid abscess.

Of the three cases of brain abscess, one has just been reported; the other two were not operated upon. In one a post mortem was made; in the other, the diagnosis was only "the most probable" from the symptoms.

Case 1.—F. F., æt. 15. Otorrhea in left ear since childhood. Treated from time to time, when the discharge was more abundant, at different dispensaries. Occasionally headaches, which became gradually more constant and violent for the last six months.

After a visit to the dispensary, where "they stopped the discharge at once," as the mother expressed it, there was a sudden violent headache, accompanied by nausea and dizziness. Next day, some convulsions, which induced them finally to call in their family physician. He found a temperature of over 104° F., slight delirium, restlessness, severe headache, and made a diagnosis of commencing meningitis. When seen by me the next day, the general condition was about the same as described. The external canal was filled with thick, fetid pus, the wall so swollen, that it was impossible to introduce the smallest speculum. The mastoid region considerably swollen, red, and tender on pressure. Mastoid operation advised under the supposition that a meningeal process had started from there, and might possibly not have advanced too far.

Operation next day. A large mastoid abscess and destruction of tegmen tympani found. For the next few days the patient seemed to

improve; the headache disappeared almost completely, the fever went down to 101, she became more conscious, answered questions. The discharge continued a profuse one through the mastoid wound as well as the external canal. Then a relapse took place, she sank more and more into coma, paralysis of the right side appeared and she died, in a comatose state, 13 days after the operation. Facial paralysis or palsy of one of the motor-oculi nerves was never noticed.

The autopsy was held the next morning. The skull was opened in the usual way; dura mater of the convexity somewhat congested, but not more adherent to the bone than usual. The veins all over-filled. On attempting to take the brain out of the skull, there was a gush of thick, yellow, fetid matter, which came out of an abscess cavity in the left hemisphere. This will be described later on. It had opened at a place opposite the posterior wall of the petrous bone, and had been adherent to its periosteum quite firmly around the point of opening; adhesions were found nowhere else. The whole dura mater of the base was covered with pus, but the greater portion of it came evidently from the abscess. After the brain had been taken out, the whole temporal bone was removed by two uniting sections.

The dura mater covering the tegmen tympani and the posterior surface of the petrous bone was thickened, and of a dirty greenish-black color. In the midst of this, over the aqueductus vestibuli, there was a small hole occluded by a protruding clot of thickened pus, which corresponded to the opening into the brain abscess. The temporal bone was then divided to show the tympanum, mastoid antrum and labyrinth. They were found to be united into one irregular cavity, partly filled with matter and detritus. A probe introduced into the wound appeared without difficulty in the middle ear. The malleus and a large portion of the thickened drum were *in situ*. The perforation of the latter was a relatively small one, in the anterior lower region. The whole tegmen tympani was carious, very thin, and showed a number of small perforations, through which pus was escaping.

The abscess cavity in the left hemisphere was situated in the temporal lobe extending backwards into the occipital lobe. It was about as large as a goose egg, of an irregular oblong shape. There was a firm enclosing capsule of connective tissue. The opening at the above mentioned place was large enough to introduce a penholder, partly, of course, artificially made. The pus within the cavity was of the most offensive character, so that it was nearly impossible to remove the odor from the hands.

Case 46.—A. B., æt. 23. Had been treated for "neurasthenia" for some time and once had to be sent away from home. After her return, was considerably improved, but always was a little "nervous."

Chronic otorrhea in right ear for four years. Suppuration ceased at intervals. Subacute attacks for 5 to 6 weeks. Was confined to bed for one week. Violent pain in whole side of head. Sensorium somewhat benumbed for one day.

When seen the following day, patient was conscious, but there was slow cerebation. She answered questions correctly, but hesitatingly. Complained of violent pain in right mastoid and whole side of head. Temperature normal, pulse 60. Swelling upon mastoid, and severe pain on pressure. Palpation anywhere on that side of the head very painful, but no spot of special localization. During severe attacks of pain, some spasms of the facial muscles could be noticed, but there was no paralysis or paresis of the facial or of the ocular muscles. The pupil of the right eye reacted somewhat more sluggishly than the left. Fundi normal.

Upon the basis of these symptoms, the possibility of an intracranial complication was considered, but no positive diagnosis arrived at, and it was decided to explore first the mastoid and await further developments.

Operation next morning. External layer of bone four lines thick; outer cells as well as antrum filled with pus. Tegmen antri and tympani carefully examined but no disintegration found. After the operation the pulse sank to 54, which symptom argued strongly for a brain abscess. The parents were informed that possibly another operation would be necessary; but declared that they would never consent to that.

During the next week considerable improvement took place. Patient was perfectly rational and the cerebral response was a fairly rapid one. Only once she complained of double vision, but when tested, no double pictures could be elicited. Temperature ranged between 98° and 99° F. The pulse rose to 74 on the day after the operation and varied then between 74 and 84. After the first week, the patient was left in the care of the family physician, and I did not hear of her until after her death.

Of the further course, Dr. K. reported:—"Patient continued to improve slowly, wound healing normally. Now and then slight headaches. She sat up four to five hours daily. Was rational. Temperature 99½° to 100° F., pulse 72 to 78. On the fifth day, she complained of severe supra-orbital neuralgia on the left side. On the sixth day, she felt weak and complained of acute headache. When I dressed wound at 2 p. m., she was perfectly conscious. In

the afternoon, a friend called and the patient was talking to her while sitting in a chair. Suddenly she experienced a terrible headache, sank back, was seized by convulsions of the entire body, the respiration became stertorous, and death followed in fifteen minutes."

Of the four cases diagnosed as meningitis, one was confirmed by the post-mortem. In the others, this diagnosis was given as "the most probable one" from the symptoms.

Case 9.—E. H., æt. 22. Severe acute otitis media in right ear after influenza. Spontaneous rupture of drum followed after a few days by cessation of pain. Same process in left ear one week afterwards. On account of considerable discharge in both ears, she consulted a physician. He prescribed insufflation of boric acid powder by the patient, twice daily. Soon afterwards, headaches and pain in the right mastoid region commenced, which increased steadily.

Four weeks after the onset they sent for another physician, who found the external auditory canal completely filled with boric acid and telephoned for a consultation at once on account of the urgent general symptoms. We found the patient very weak, temperature 102° F., pulse 130, small. Both pupils dilated nearly *ad maximum*, with very slight reaction. Choked disc in both eyes, more pronounced in right than left. Ptosis of left upper lid. There was, in addition, most probably a paresis of one of the external eye muscles, but the comatose condition of the patient made an exact diagnosis impossible. Hardly any swelling in mastoid regions. After the removal of the boric acid from the external canal, there was a large perforation found in each tympanic membrane and a small amount of pus in the tympanic cavities. No swelling of the walls of the external canals. We arrived at the diagnosis of purulent meningitis, the path of infection being most probably directly through the tegmen tympani, and I declined operative interference, as I considered the case too far gone.

During the next two days the symptoms increased in severity, but on the morning of the third day there was a marked improvement. The sensorium had become quite free, and there was a large swelling under the fascia of the sterno-cleido of the right side, due to a sinking abscess. The patient herself remarked that "it had gone down there now." Dr. S. called me in again, and we decided that the case might not be beyond operative reach. Operation same day. After the first strokes with the gouge, the external auditory canal filled with thick, yellow pus. In the bone, a large abscess cavity was found. The superior and mesial wall was carious, and an opening through it lead into another abscess cavity. Whether this latter was subdural or intradural, I am unable to state, for at the same moment

respiration ceased and a few minutes afterwards she died on the operation table. Post mortem not permitted. Whether this patient could have been saved by operating after the first visit, remains an open question. The palsies spoke certainly more in favor of a diffuse meningitic process.

Case 60.—J. S., col., æt. 48. Otitis media and otorrhea in left ear for two months. Typical mastoid symptoms for two weeks. Operation emptied a large abscess and detritus cavity, reaching into antrum. The course was an absolutely normal one for three and one-half weeks, and not the least symptom suspicious of an intracranial affection was manifested. The patient came to the clinic for the dressing from the sixth day after the operation. On the 26th day, he had a chill and headache and vomiting. Confined to bed two days. There was suspicion of sinus thrombosis, but the attack seemed to have been one of gastritis, after eating too many tomatoes, and passed over entirely. The suppuration from the ear had ceased in the third week, and the mastoid wound had closed and the patient was dismissed six weeks after the operation.

The next day, he came back in a half comatose condition and stated that he could not pass urine for the last 24 hours. Sent to City Hospital. The catheter found only a small quantity of urine in the bladder. Patient became more comatose, pulse 72, temperature 104.5° *in ano*. The differential diagnosis between uremia and intracranial complication was left open. I intended to make an explorative craniotomy next day, but the patient had regained consciousness, temperature had fallen to 99° F., and there was only some occipital headache. During the next few days he could not be seen regularly on account of extraneous reasons. He passed again more and more into coma, temperature ranging from 101° to 103° F. He was very low on the fifth hospital day. Second operation, wound reopened, mastoid filled out with healthy cicatricial tissue. Wall of sinus firm and hard, therefore no further investigation in this direction, but trephining in cerebellar region, because the headache was most pronounced in the occiput. Some dirty lymph and small amount of pus found inside of dura. No cerebellar abscess. Patient died two days later without regaining consciousness.

Autopsy. Diffuse cerebro-spinal meningitis, with tubercular nodules. Infiltration of pia thickest in right side, over frontal lobes, and below cerebellum and medulla oblongata. Dura covering temporal bone everywhere intact. Sinus opened; contained fresh blood clot. The temporal bone was removed: Entire tip of petrous pyramid was wanting, no cochlea or semi-circular canals present. The destruction was evidently due to a tubercular process, the remnants being covered with cheesy masses and tubercular nodules.

In all probability, the caries of the petrous pyramid was the primary focus, which affected the mastoid and middle ear secondarily. It is noteworthy, that the latter two healed, while the chief lesion, which was of course beyond operative interference, gave rise to the diffuse tubercular meningitis.

Case 65.—J. D., æt. 4. When seen in first consultation, the child was in a half comatose condition. Slight mucous discharge from right ear. No swelling or redness on mastoid. I advised, for one or two days, expectant treatment, but was not called again until about one week afterwards. There was now a slight swelling in mastoid region, the child more comatose. The temperature was 103° to 104° F. *in ano*, but not of pyemic type. Next day operation. Cheesy pus in antrum. Sinus wall discolored. Sinus opened. Infective thrombus removed. Jugular vein ligated.

Picture did not change much after the operation. Temperature 104° to 105° F. Convulsions and increasing coma till fatal end four days after operation. The child was of a scrofulous type, and there is tuberculosis in the family.

Case 98.—H. S., æt. 9. Chronic otorrhea with fetid discharge for years; never any treatment. Suddenly became sick about four days ago, with high temperature and severe headaches. Attending physician, after excluding acute infectious diseases, diagnosed an ear complication and sent for me. Temperature was then 105° F. Patient half comatose, lying on left side, uttering cries at intervals. Could be aroused by talking to her, and answered slowly but correctly. Facial paresis on left side. Orbicularis only moderately paralyzed, while all muscles controlled by lower branch of facial, were completely paralyzed. No other paralysis or paresis. No swelling nor redness on mastoid or emissary regions; pain on pressure very doubtful.

Diagnosis was made of intracranial complication; furthermore, that the process went directly through the tegmen tympani without mastoid involvement; that the process was in all probabilities a diffuse meningitis, but that an abscess in the temporo-sphenoidal lobe could not be excluded with certainty. Therefore, as a last chance to save life, it was proposed to make an exploratory operation.

Operation next day. Mastoid opened in the usual manner, found healthy. Then trephined one cm. above external auditory meatus. Dura not bulging, brain substance edematous. Explored temporo-sphenoidal lobe in different directions without finding pus. Child remained in an unconscious condition until death next day. Necropsy not allowed.

I do not deem it necessary to add many remarks concerning these cases, after all that has been written of late on the subject of intracranial complications. Some of them show, that it is still exceedingly difficult, if not impossible, to make a differential diagnosis between them.

THE PRESENT STATUS OF THE TREATMENT OF MASTOIDITIS.

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Mastoiditis and its treatment have been for the past thirteen or fourteen years a fruitful theme for thoughtful discussion. Numerous articles from able pens have been contributed and we now pause and contemplate what the present status of the treatment of the disease is. In order to arrive at some conclusions, the essayist addressed some questions to leading otologists of this country and Canada. Numerous replies have been received which are tabulated in the table herewith submitted. A great number of the replies received do not reveal the writers' names. By exclusion the essayist thinks that he has located some but others he could not, and only those names appear in the table who signed their answers and those whom the writer feels sent answers. The questions submitted are as follows:

- (1) Your treatment for acute mastoiditis, especially concerning the application of heat and cold or both to the mastoid process?
- (2) How long do you apply cold to the mastoid before resorting to operation or heat?
- (3) If you use heat, how do you apply it?
- (4) Do you make an exploratory operation for mastoiditis?
- (5) In what proportion of your cases of mastoiditis have you found operation necessary?

It is but fair before submitting the answers to these questions that I give the results of my own experience.

In acute mastoiditis my preference has always been for dry heat. I believe that according to the recognized principles of surgery that in such cases we want diapedesis and not stasis. To accomplish this end we need an increase in the size of the blood vessels to more readily remove the obnoxious products of inflammation, and as Nancrede well says in his "Principles of Surgery," "The only means which will produce this local increase in the size of the veins is heat, and a moderate degree of this, such as can be comfortably endured." The heat relieves the pain and does not mask the symptoms. Cold

may do the first, but it does mask important symptoms. Cold will diminish the caliber of the vessels, but the vitality of the cells are impaired and the leucocyte's activity is diminished. Hence stasis and thrombosis of the vessels at the seat of inflammation. (Nancrede.)

A case reported to the section of Laryngology and Otology of the American Medical Association at its Saratoga meeting shows the bad effect of long continued application of cold. It had been applied continuously for one month, the patient still suffering pain, but without tenderness over the mastoid when examined for the first time by the writer. Yet when operated upon the whole process was necrotic, the tegmen eroded and a subsequent operation revealed a large brain abscess in the temporo-sphenoidal lobe which had been missed in the first operation, though searched for because of the symptoms of brain abscess.

Many who use cold externally also use hot douches frequently repeated within the external auditory canal—a fairly good compromise. In the application of the heat no better means of its application can be found than in the little Japanese pocket stove. It is light, will burn for two to three hours at a constant temperature, and can be closely applied to the head of our patient by a bandage. This cannot be said of the hot water bottle or the bran bags. The amount of heat passing out into the tissues can be nicely regulated by wrapping it with a handkerchief or gauze. We cannot expect much of result by using the hot water bottle or the Leiter coil. We must have something which can be closely applied to the head and which will keep a constant temperature for hours at a time.

As Dench in a recent article points out an exploratory operation is not amiss when needed to clear up a diagnosis. The mastoid operation is simple and it is rarely the misfortune of a competent surgeon to lose a case from operation for simple mastoiditis.

It is the experience of the writer that operation is necessary only in about one out of eight cases, using the treatment outlined above together with such general remedies as any case may call for, e. g., blood letting, paracentesis of the drum, etc. Having thus briefly given his views, the writer will briefly pass over the view of authorities who have not answered these questions, but whose answers may be found in their writings.

In the *American Journal of Medical Sciences*, April, 1902, B. A. Randall believes that in the abortive treatment of acute mastoiditis heat is superior to cold.

Alderton in Posey & Wright's new work on "Diseases of the Eye, Nose, Throat and Ear," advocates the douching of the external

auditory canal with hot water and the external application of heat to the mastoid process.

Politzer in his late work on "Diseases of the Ear" advocates cold.

Dench advocates heat in preference to cold.

Barr prefers heat.

Roosa in his work, published in 1885, prefers poultices.

Burnett in "Diseases of Ear, Nose and Throat" by Barnett, Ingalls and Newcomb evidently believes in immediate operative interference. (P. 168.)

Clarence J. Blake in "Burnett's System of Diseases of the Ear, Nose, and Throat" (1893), advocates the use of cold over the mastoid together with paracentesis of the drum membrane.

McBride in "Diseases of the Throat, Nose and Ear" (1894), advocates the use of cold.

- Bishop in his book advocates the local application of cold.

Horace G. Miller in "American Text Book of Diseases of the Eye, Ear, Nose and Throat," advocates the use of dry heat.

Pritchard's "Diseases of the Ear," recommends the avoidance of cold applications and in "deep mastoiditis" the use of hot applications with the other treatment which he recommends.

Bacon in his "Manual of Otology" recommends cold in early stages.

In his work on "Diseases of the Ear" (1886), Pomeroy gives preference to poultices.

In mastoid abscesses and their treatment Broca and Lubet-Borbon recommend cold. Sargeant F. Snow is a firm believer in the very prolonged application of cold to the mastoid process, but also recommends hot water injections by a fountain syringe. Mark Hovell prefers cold by the means of the Leiter coil.

NAME.	QUESTION I.	QUESTION II.	QUESTION III.	QUESTION IV.	QUESTION V.
Atkinson, J. W. Paerson, N. J.	Heat (dry). Bichloride douches (hot) of auditory canal after paracentesis.		Hot water bags or bran bags.	Only when symptoms indicate infection beyond middle ear.	
Ayres, S. C. Cincinnati, Ohio.	Heat.	For a few days.	Japanese pocket stove.	No.	Large proportion.
Barnhill, J. F., Ind.	Cold.		Hot water bottle with in- descent bulb over it.		
	Heat where cold is intolerable. Paracentesis	48 hours.	Japanese pocket stove.		
Booth, R. S. Troy, N. J.	Cold at start. Heat if not relieved by cold.	24 to 48 hours.	Hot water bag.	Not often.	Large proportion.
Brace, L. D. Evansville, Ind.	Cold, if not relieved in several hours—Heat.	48 hours.	Hot water bag. Salt bag.	No.	Large proportion.
Brown, Price. Toronto, Can.	Heat.		Bran bag.	No.	50 per cent.
Bulson, A. E., Ind.	Paracentesis.	24 hours seldom.		No.	50 per cent.
Carow, E. C., Pittsburg, Pa.	Cold.	48 hours.		No.	95 per cent.
Calloun, A. W., Ann Arbor, Mich.	Heat (preference).	No definite time.	Hot water bag. Salt bag.	No.	20 per cent.
Chambers, T. R., Jersey City, N. J.	Cold. Paracentesis. Cold for 20 minutes. Heat for 40 minutes if symptoms are violent.			No.	
	Hot douches.			No.	15 per cent.
Chentam, W., Louisville, Ky.	Heat.	24 to 48 hours.	Flax seed poultices.	No.	None.
Christy, G. C., Pittsburg, Pa.	Cold seldom.			No.	
Cline, L. C., Indianapolis, Ind.	Heat.			No.	
Clough, H. T., Bangor, Maine.	Cold (6 to 12 hours). Dry heat.	12 hours.	Commercial bags. Hot compresses.	Yes.	90 per cent.
Connell, J. C., Kingston, Ont.	Dry heat.		Hot water bottle.	Yes.	Proportion small.
Crouch, J. F., Baltimore, Md.	Heat.	24 hours.	Hot compresses.	Yes.	
Davey, J. R., Chicago, Ill.	Cold.	24 to 72 hours.	Don't use heat unless cold is intolerable.	Yes.	10 per cent.
Day, E. W., Pittsburg, Pa.	Heat and cold. Change of either anti-phlogistine.	24 to 48 hours.		Yes.	80 per cent.

NAME.	QUESTION I.	QUESTION II.	QUESTION III.	QUESTION IV.	QUESTION V.
Dudley, W. H., Easton, Pa.	Cold.	24 to 36 hours.		No.	70 to 80 per cent.
Dunn, J., Richmond, Pa.	Heat.		Irrigation by hot water.	No.	All.
Ellis, H. Bert., Los Angeles, Cal.	Heat.		Hot water bag.	No.	
Farrell, T. H., Utica, N. Y.	Paracetanis.	48 hours.	Japanese pocket stove.		50 per cent.
Grady, L. B., Nashville, Tenn.	Heat by irrigation.		By irrigation.	No.	
Harlan, Herbert, Baltimore, Md.	Heat and Cold.		Hot water bottle.	Yes.	50 per cent.
Holmes, C. R., Cincinnati, Ohio.	Purgation.		Japanese pocket stove.	Should not hesitate.	Very large proportion.
Holt, E. E., Portland, Maine.	Pilocarpine.		Hot alkaline solutions every two hours.	Seldom.	All.
Hubbard, Thos., Toledo, O.	Heat.	If used at all only for short time.	Dry and moist.		
Ingersoll, J. M., Cleveland, Ohio.	Heat.	If used, not longer than 24 hours.	Hot water bag.	Rarely operate.	
Jackson, C., Pittsburg, Pa.	Heat after 24 hours and no change is apparent.		Leiter coil or bag.	No.	90 per cent.
Johnson, W. B., Patterson, N. J.	Cold.	48 hours.		Yes.	
King, Gordon, New Orleans, La.	Rest in bed. Catharsis.			No.	10 per cent.
Koyle, F. H., Howellsville, N. Y.	Heat.	24 hours.	Japanese pocket stove.	Yes.	75 per cent.
Kyle, J. J., Indianapolis, Ind.	Cold.	24 hours.	Hot water bag.	No.	None.
Landfried, C. L., New Orleans, La.	Heat after 52 hours of gradual improvement.	48 hours.		No.	All cases early.
Lewy, Rob't, Colo.	Heat.		Flaxseed poultices.	No.	Extremely large proportion.
Libby, Geo. F., Colorado Springs, Colo.	Cold.	48 hours.		No.	30 per cent.
Lincoln, Wm., Cleveland, Ohio.	Cold for 24 to 36 hours. Heat after this.	12 to 36 hours.	Hot water bag.	Only once.	10 per cent.
	Heat.			No.	

NAME.	QUESTION I.	QUESTION II.	QUESTION III.	QUESTION IV.	QUESTION V.
McCoy, Jas., New York, N. Y.	Cold (in acute non-suppurative cases). Heat (in hemorrhagic cases following grippé). Cold. Paracetesis. Purgation.	36 to 48 hours. 24 hours.	Hot douching of ext. auditory canal every 2 or 5 hours.	Yes.	80 to 85 per cent.
McKernon, J. F., Y. Watertown, N. Y.	Heat.	24 hours.	No.	No.	3 1-8 per cent of acute cases.
McReynolds, J. O., Dallas, Texas.	Heat.		Hot water bag. Moist cloths with hot water bottle on top.	Not often.	Very seldom.
Martin, W. A., San Francisco, Cal.	Heat. Heat, generally. Cold, seldom.		Poultice. Hot douches in external auditory canal.	Yes.	75 to 85 per cent.
Moss, R. E., San Antonio, Texas.	Heat.		Hot water bag.	No.	50 per cent.
Munger, C. E., Waterbury, Conn.	Cold 24 hours. Heat.	24 hours.	Flaxseed poultices.	Yes.	
Murray, G. D., Scranton, Pa.	Heat.	24 hours.	No.	No.	75 per cent.
O'Kelley, J. P., New Orleans, La.	Cold.		Dry heat.	Yes.	
Parker, E. F., Charleston, S. C.	Dry heat.		Hot poultices.	No.	
Phillips, W. C., New York, N. Y.	Heat. Cold.	24 to 36 hours.	Bags or coil.	No.	50 per cent.
Powell, S. C., Newport, R. I.	Heat at first. Cold later if heat does not improve.	Few hours.	Poultices.		
Ray, J. M., Louisville, Ky.	Heat.		Hot water bag.	No.	50 per cent.
Reynolds, Dudley, Louisville, Ky.	Cold. Heat occasionally.	24 to 48 hours.		No.	66 per cent.
Richards, Geo. L., Fall River, Mass.	Heat	48 hours.	Hot douches in ear.	No.	75 per cent.
Richardson, C. W., Washington, D. C.	Cold.	24 to 48 hours.	Leiter coil. Flannel wrung out of hot water.	No.	20 per cent.
Roberts, W. H., Pasadena, Cal.	Cold first. Heat afterward.	48 to 72 hours.	Hot water bag.	Occasionally.	75 per cent.
Rogers, J. T., R. I.	Heat.	48 to 72 hours.	Hot water bag.	Yes.	10 per cent.
Smith, Harvey, Winnipeg, Man.	Cold for 24 hours. Heat afterward.	24 hours.	Hot water bag.	No.	80 per cent.
Sterrett, J. K., Pittsburg, Pa.	Heat.		Hot water bag.	No.	50 per cent.
Stucky, J. A., Lexington, Ky.	Heat.		Hot water bag.	No.	50 per cent.

NAME.	QUESTION I.	QUESTION II.	QUESTION III.	QUESTION IV.	QUESTION V.
Theisen, T. F., Albany, N. Y.	Heat.		Hot water bag.		Rarely.
Tiffany, F. B., Kansas City, Mo.	Heat occasionally.		Electric coil.	No.	
Toeplitz, D. W., New York, N. Y.	Heat after 48 hours.	48 hours.	Poultices. Hot bags.	No.	25 per cent.
Wells, W. A., Washington, D. C.	Cold. Heat after 72 to 96 hours.	72 to 96 hours. Then heat.	Moist Compresses.	No.	
Wheelock, K. K., Mt. Wayne, Ind.	Heat.		Water bag.	Yes.	
Willets, J. F., Pittsburg, Pa.	Heat, but pref. early operation.			No.	
Wilson, N. L., Elizabeth, N. J.	Heat in ear and canal.	36 hours.		No.	"
Winslow, J. R., Baltimore, Md.	Cold over mastoid. Cold if seen early. Heat otherwise.	12 hours.	Hot water bag. Japanese pocket stove. Hot bavs. bag. Hot water bag. Leiter coil.	Rarely.	33 1-3 per cent.
Wishart, Toronto, Can.	Heat.			Yes.	20 per cent.

DISCUSSION.

DR. J. W. MURPHY (Cincinnati).—In reference to Dr. Keiper's paper as to whether we should use heat or cold in the early stages, we have good authorities on both sides. If I see the case at a very early stage I am partial to the ice cap, but never for more than 24 hours. If there is no improvement at the end of 24 hours, I then go to the other extreme, and use heat. I much prefer dry heat to moist, and recently I have been able to apply the dry heat quite conveniently by means of the electric globes whenever in the hospital or the home we have the electrical current. I use one of these drop lights on a cord, with a key on it, so the current can be controlled by the patient. I wrap the globe in a towel and place beside the ear within the control of the patient. As it gets too hot it can be turned out. It must be watched and the patient not allowed to go to sleep, as these get very hot; I find a napkin can be scorched and almost burned by the heat from one of these globes. I also like to douche these ears with very hot water. I like the Lucae ear douche in which a return current is allowed to escape from the ear. It is attached to the fountain syringe and the little hard rubber tube placed in the ear so the current of hot water gives the most satisfactory results. The patient can apply this himself. The symptoms of mastoiditis must be carefully watched, and as the doctor said, cold is apt to mask dangerous symptoms; therefore if the symptoms do not improve in a short time the cold should not be persisted in. I have seen during the past winter a number of cases, resulting from the recent epidemic of grippe. It has seemed to have a special tendency to involve the middle ear. Last week I had a case of this kind, in which cold was applied without beneficial results; then I tried heat. The patient never had a temperature to exceed 99 degrees, and deceived me as to his reports each morning, saying he had rested well and was getting along nicely. We all hate to do a mastoid operation if there is any hope otherwise, and I took the patient's statement; but a few days later I noticed a peculiar bulging of the posterior superior wall of the canal and refused longer to be responsible for the case, telling him I was sure he ought to have the mastoid opened. He consented to the operation and I found an abscess at the tip of the mastoid in contact with the lateral sinus. The abscess was larger than a pigeon's egg and full of offensive pus, and yet the man had few symptoms of mastoiditis, which shows how often we can be deceived.

The cases reported by Dr. Barck were interesting and show the difficulties we frequently encounter in being unable to diagnose the sub or extra-dural abscess in these cases, especially in children. In

the past six weeks I had a case that demonstrated this clearly. The child had an attack of grippe and there were very few symptoms; no discharge from the ear, but a constant complaint of pain. A paracentesis was made and a drop of bloody serum exuded. Several days later the mastoid was opened and a small amount of granular tissue, in a comparatively healthy ear, was removed. It was decided in consultation that probably there was some inflammation of the brain, and the wound was opened up and the integument removed. No abscess was found. Several days later the symptoms still pointed so strongly to a brain lesion that the wound was again opened by a general surgeon in consultation and further exploration revealed no abscess. Meningitis resulted fatally a few days later.

DR. GOLDSTEIN.—There is very little in either paper that will bear discussion, one being a synopsis to date and the other simply statistical data; but the question developed by Dr. Murphy, the comparative value of thermal applications is interesting, and I think it is just as much in controversy to-day as it was five or ten years ago. Just as the general surgeon in his treatment of inflammations see-saws back and forth between heat and cold, so too, does the otologist. I am glad to endorse Dr. Murphy's suggestions in the case which he has related, which is of the class we see so much of now, the acute grippe inflammations of the ear, in which the actual pathological inroads, brought to our notice when the mastoid is opened, occur with remarkable rapidity. Neither heat nor cold will benefit a case where mastoid symptoms develop a day or two after an acute ear ache following influenza. You will find granulations almost filling the antrum. How heat or cold or other agent will relieve that condition I fail to see.

I would like to ask Dr. Barck in regard to one case to which he refers which has an interesting history—that of the recurring mastoid operation. At about what time was that done? Was it done within the time of the recent radical operation? (Dr. Barck: In 1892-94-95.)

Unless there was a distinctly tubercular or other equally progressive specific infection in that mastoid, there should not have been an occasion for such frequent operating.

DR. G. W. SPOHN (Elkhart, Ind.).—I was very much interested in both papers; but one thing not mentioned was the use of electricity. I tried it once in a case for operation, where the consent of the family could not be obtained. The ear bulged out and was in an edematous condition in the region of the mastoid. At the fifth and sixth days the temperature was $101\frac{1}{2}$ and 102. I used the high frequency current for fifteen minutes lightly over the mastoid, for two

days in succession. To my surprise, after the second application there was great improvement. In four days the fever had disappeared and the case made a good recovery. I applied the current to relieve the pain, as I had done previously in cases of a neurotic origin.

I have had old chronic cases of otorrhœa that were carefully treated for many months with no apparent improvement, which were cured with the application of the X-Ray. It seems to me the old mastoid sinuses, left after operation, could be more speedily cured with the X-Ray. It has a stimulating action that is wonderful in pus discharging sinuses. The ray is used very successfully in adominal sinuses by such renowned surgeons as Murphy, Senn and many others.

DR. BRADFIELD.—I would like to ask about bleeding subsequent to an acute otitis media; what is the experience as to the benefit derived from free hemorrhage?

DR. L. C. CLINE (Indianapolis, Ind.).—I wish to speak a word in reference to the heat. The question reminds me of the bicycle fad we had a few years ago; if you were lean you should ride the wheel to become fat; and if you were fat you should ride to become thin. This is the case with the hot application. It is all right to use heat, but I doubt its efficacy in a great many cases. We have got to do something if we have mastoid involvement, or if we are threatened with it. I think if we would turn our attention to flushing the alimentary canal we would do better than by applying cold or heat. A little calomel followed by Rochelle salts or anything to clean out the tract and put the house in order. If it is a surgical case, nothing will do good but surgery. If the pus has formed in the mastoid cells I do not believe that either heat or cold will stop it. I believe the internal treatment will come nearer doing it than hot or cold applications. The point is to find out when we should operate, and then operate without further delay. Many cases die from neglect to operate soon enough. When we find the bulging of the posterior superior wall we should become suspicious, and as Dr. Murphy said, we should insist on the operation or dismiss the case.

DR. W. R. MURRAY (Minneapolis).—Referring to Dr. Keiper's paper, I do not believe there is any therapeutic effect but the relief of pain in the ice pack. It is grateful to the patient, but it is capable of masking a great many symptoms and I do not think it has much effect on the mastoid disease. It should not be applied for more than 24 hours.

I never saw a case clear up after there was bulging of the super-

ior and posterior walls of the canal. When that occurs, the indication is absolutely for operation.

Referring to Dr. Barck's paper, which has been very interesting, I would like to ask whether, in his cases of sinus thrombosis, it is always his policy to ligate the jugular vein before going into the sinus. I would also ask his policy in the after-treatment for brain abscess in the way of dressings and the frequency with which they are made.

DR. BARCK (closing).—As regards the relative merits of heat and cold, I believe this question is still an open one in medicine as well as in surgery. Personally, I prefer cold applications in children and in young robust people. In middle aged and old persons, I prefer warm or hot applications, usually by the dry method. I consider, however, this point of minor importance. In my opinion, the best remedy to abort an acute mastoiditis is an early paracentesis. By this procedure, certainly a number of them may be prevented. But there are instances where neither a paracentesis nor any form of application can prevent mastoid affection. The cause lies in the peculiar anatomical condition of the temporal bone. I have examined a number of these as to the relative position of the antrum and the middle ear. While in some of them, the floor of the antrum was just as high as the opening of the aditus into the middle ear, or even slightly higher, in others it was decidedly lower. In the former, there is a relatively free communication between the antrum and the middle ear and so even after the infection had reached the antrum, there is an outlet for the secretion. In the latter, there is a kind of cul-de-sac, and when this is affected, it will in all probabilities not recede, no matter what kind of external application is made.

As for bleeding by leeches or other means, I use it quite frequently in well nourished individuals, and believe it acted well in a number of instances.

In answer to Dr. Goldstein, as to the case, which was operated upon four times, will say, that there was never an indication to perform a radical operation, because there was no chronic suppuration from the middle ear. It was in every instance an acute otitis and acute mastoiditis—in fact the most acute and rapid one I ever saw. After each operation, the otorrhea ceased, the perforation of the tympanic membrane closed, and the hearing returned about to the normal. It was so a year after the third operation, as stated. I never saw any reason to sacrifice the chain of healthy ossicles, and with them the hearing. I simply reported the case, which was certainly unique and is still an enigma to me in many points and as to its etiology.

In regard to the drainage in brain abscess, would like to state that I believe in frequent dressings. After the operation I usually allow two days to elapse, and then institute daily dressings.

PROGRESS IN OTOTOLOGY IN FIFTY YEARS.

BY FAYETTE C. EWING, M.D., ST. LOUIS.

Some of you, for a moment, may be tempted to draw a long breath over the title of this paper, but there need be no cause for alarm over its possible length. Fifty years have accomplished wonders in medical and surgical science, and the ear is but a small part of the human machine and the catalogue of its progress may be run in the time of my allotment. Of course, were I to attempt a complete record of the history of otology for the past fifty years, detailing and describing the progress, step by step, the course by, or through, which, each conclusion was arrived at, and dwelling upon the minutiae of pathology, histology, bacteriology, and medicinal and operative therapy, I should very likely exceed the limit of time. However, the title of my paper admits of a much more liberal and cursory treatment of my subject, and I shall do no more than attempt a synoptical historical sketch; a mere outline reflecting the progress of otology for the past half century.

I confess that when I handed in the subject of this paper to your secretary I had expected that it would require more time for its enumeration than I shall allow it, the error growing out of my restriction to more recent discovery, some of the approved methods of modern otologists. The practitioner of to-day is one who seeks new, instead of old knowledge and wishes to be "up-to-date," hence, he buys new editions and rids himself of the old. He wants results, and in a progressive science like medicine the physician is required to be on the alert that he may be equipped to offer the most potent remedies for human suffering, and that he may not be placed in contrast with his neighboring competitor to his discomfiture and humiliation. So while I might insult some of you if I offered a paper attempting to instruct you on "up-to-date" otology, I can, without fear of offense, draw a dividing line between the knowledge of the otologist of 1853 and that of us of 1903, enabling some of you, no doubt, to appreciate the fact that much of our vast present day attainment was the possession of the otologist of fifty years ago. It happens that I have for reference the standard work of that day as a line of demarkation separating what they knew from what we know. It serves my purpose, and in what

I shall say, William R. Wilde's "Practical Observations on Aural Surgery and the Nature and Treatment of Diseases of the Ear," 1853, is my chief authority.

While I do not minimize the importance of the scientific knowledge which we possess of the ear and its diseases, I make bold to say that the most important discovery which we have made since 1853 has been in the elaboration of the work of the surgeons and not through the individual discoveries of otologists or through disassociated achievements. Our progress is largely embraced in the treatment of suppurative conditions in which the elements of pathology, histology, bacteriology, instrumentation and operative technique have part; all of which are more or less connected with surgical procedure than general medical therapy.

In pathology we have the basis of the whole scientific fabric, and in his application of it to causes and effects, the otologist has been no laggard, but has caught the spirit of all medical science. Without a correct knowledge of pathology we are in the dark and incapable of intelligently addressing our therapeutic resources.

Now, while the aurist of the early half of the past century knew the anatomy of the ear as minutely as we do, he did not appreciate the intimate relationship and bearing of one organ upon another, nor the etiological influence of the one as a factor in the production of disease in its neighbor. For instance, he was not cognizant of the effect of nasal upon aural parts, as influential in the production and continuance of disease in the post-nasal space, and its extension through the eustachian tube into the middle ear. We made a long step when we arrived at an appreciation of the necessity for free nasal passages and a healthy mucus membrane as indispensable to the cure of certain conditions within the tympanum. To one man do we owe the one great discovery of otology for the advancement of our science, the only discovery of unquestionable and consistent value made by otologists during the fifty years past. Need I mention Wilhelm Meyer? I confine myself to contributions of those of our cult, and restrict their offerings to those that were not built upon the foundations of their predecessors, nor are modifications, however meritorious, of methods already promulgated. To Lister, surgeon, do we owe the major part of our progress in scientific otology from the time I have elected to consider, for within this great epoch has been developed the perfection of all surgical procedure to which otology owes most of the commanding basis it now occupies. Wilde and his contemporaries knew the importance of antiphlogistics in acute congestion in the parts adjacent to the middle ear, and they fully appreciated the

value of drainage and cleanliness as demonstrated in "Wilde's Incisions," but without antisepsis they had not learned with what impunity the human body may be penetrated and how thoroughly the mastoid may be drained. Our appreciation of the tremendous importance of pus elimination has come to us through the teachings of Lister, the influence of antisepsis, otology having developed in the same way that it has affected all surgical consideration.

In instrumentation, diagnostic and operative, we have first in importance the laryngoscopic head mirror, through which our explorations of the ear is more complete. Our tests and discernments of ear diseases are far more comprehensive and accurate, based as they are upon increased knowledge of histology, bacteriology and pathology. The use of the tuning fork, Galton's whistle, Seigle's speculum, otoscope, etc., have all perfected us in diagnosis, while the impunity of surgery has naturally added boldness to dexterity. In this connection it is meet that I should refer to the Politzer bag, the most valuable adjunct to the aurist's armamentarium in diagnosis and treatment of congestive conditions in the eustachian tubes and middle ear. Notwithstanding its inferior importance, to Meyer's elucidation of adenoids and the still greater revelation of antisepsis in estimating the value of Politzerization, so called, it were well to remember that the catheter and Valsalva's method antedated it many years, and the Politzer discovery does not involve a demonstration of the improvement through inflation of the middle ear in Eustachian tube catarrh. The fact was well known by the aurists of the time of Wilde and before, and Politzer simply demonstrated a new method built upon the old, by which air may be forced through the Eustachian tubes into the middle ear cavity. It was, indeed, an improvement, and a more scientific method than Valsalva's, but when we consider that all this was well known, and the ease with which the ear may be inflated by means of these several methods, the mystery is not that Politzer discovered inflation by a rubber bag, but how it happened that it was not discovered by some of his predecessors. In the diagnosis and treatment of tubal catarrh and in certain nasal obstructions, Politzer's bag is indispensable, but in the large majority of cases the catheter is far more effective and scientific. Catheterization addresses a direct and concentrated stream of air, vapor or fluid through the tube into the tympanum, while the bag projects a distributed and rarified current of air or vapor only. The modifications of Politzer's method are much more satisfactory than the simple bag for office uses, though the latter is a valuable adjunct at the bedside.

In summing up the advances made in otology during the past fifty years I conclude that they almost entirely hinge upon antisepsis,

pathology, bacteriology, histology and operative technique. Out of these have grown perfection in diagnosis, and a clearer realization of the necessity for drainage and cleanliness in suppurative conditions and an improved operative technique together with a skill and boldness in draining cavities containing pus. Pathology and its collateral branches add to our knowledge of the cause and course of diseases and are used as methods for the arrest and eradication of offending matter. Out of the appreciation of etiology has come a more perfect perception, and, leading on, these have developed accuracy of diagnosis and skill in operation until at last we have, full fledged, a specialty recognized as one of dignity, through skill and achievements.

I would that I might dwell longer upon the list of inventions and discoveries by otologists, but however we may have arrived at our improvements and advances they are admitted and are permanent. Our least accomplishment seems to have been in the treatment of chronic deafness without suppuration, and I particularly refer to that intractable malady known as sclerotic deafness. The various operations and instruments designed to improve sclerotic deafness are all of doubtful utility and those who are treated for this disease are improved just about as much as they were fifty years ago, when some aurists poured glycerine into their ears. We have learned to distinguish between catarrhal deafness and a true sclerotic process, but in naming the disease have not named a remedy. Thus it is that this common class of sufferers go on and on until finally wearied of the ineffectualness of treatments they become a menace to our material success and a reproach to otologic science. And what has been said of sclerosis can be applied to internal ear diseases. Here again we have added a little to our diagnosis and named a disease or two without naming a remedy.

On the whole, we have done well, considering that it has been many years less than fifty since this branch of medical science attained the dignity of a distinctive science and evoked a concentrated effort of a large body of workers. Now that all of medicine is upon such a scientific basis and so many are concentrating their efforts upon otologic science entirely, which is recognized as a department of medicine of vast importance, we may expect that in 1953, should some one elect to write an account or resume of the progress of otology in the interim between now and then—as I have tried to do of the preceding half of the century—he may point to a large list of achievements by otologists, standing distinctly upon their own foundations, and not elaborations of the work of others.

REPORT OF TWO CASES OF LARYNGEAL PARALYSIS DUE TO AORTIC ANEURISM.

BY HAL FOSTER, A. B., M. D., KANSAS CITY, MO.

Laryngeal paralysis is always interesting to the physician.

This subject is very important in general medicine.

The cause in each patient should always be ascertained, when possible.

I am quite sure, a brief report of the following cases will not be without interest to this society.

November 24th, 1897, Mr. H. M. S— was referred to me by Dr. Wainright. The doctor saw the patient the day before for the first time. The patient was born in Ohio, and was 61 years old. He had never been married. His occupation was that of a retail cigar and tobacco dealer.

For many years, he walked ten blocks to his store and opened it at 6 a. m., and closed at midnight. You see he led a closely confined life. He was a tall thin man. His digestive organs had been in poor condition for years.

When he was 25 years old he contracted syphilis, but placed himself under the care of a physician for three years, at the end of which time, he was pronounced cured. He smoked three cigars a day and never indulged in liquor of any kind.

About a year before the writer saw him, he noticed that he was somewhat short of breath, and gave up walking. Occasionally a little blood would be expectorated.

A firm of advertising physicians treated him for about ten months. They told him he was suffering with catarrhal trouble and would be permanently cured in twelve months. A few days before I saw him, he lost quite a great deal of blood from coughing. It was for this condition of things he consulted Dr. Wainright.

There were no bacilli in the sputum. The patient was only able to speak in a whisper when I saw him, and was breathing with great difficulty. The least exertion added to his labored breathing. He was still raising a little blood on arising every morning.

On physical examination, the pulsation and thrill could be heard and felt. We had no difficulty in making the diagnosis as aortic aneurism. On laryngeal examination, the left vocal cord was found to be paralyzed, it was in the cadaveric position. This patient would

have a great deal of trouble breathing at night, so much so, that his sisters could hear him in the adjoining room.

His countenance was anxious. His sisters were frankly told of his exceedingly alarming condition, and we insisted on his remaining quiet at his home. He was given iodide of potash and small doses of heroin at night. This treatment relieved him so promptly, that for a time he insisted that he would recover in the spring.

December, 1897, he was presented before the Kansas City Academy of Medicine. Our diagnosis was concurred in as well as treatment. A nutritious but non-stimulating diet was ordered. I gave him mild antiseptic inhalation, which always gave him relief.

This treatment was continued until January 15, 1898. Early in the morning, January 16th, during a paroxysm of coughing, while talking to his sister, the aneurismal sac ruptured, which caused an instant but painless death.

Dr. Wainright, assisted by Mr. Wagner, myself and several students made the post-mortem, which confirmed our diagnosis. The specimen was presented before the Academy. There was some necrosis in the vertebræ, caused by constant pressure. This patient never complained of any pain. Dr. J. N. Scott took an X-Ray picture of the chest before death, and also confirmed the diagnosis.

Sir Felix Semon, says that one of the symptoms of aortic aneurism is a slight paralysis of the vocal cord. The Semon law is undoubtedly a very interesting and useful one, and physicians should remember it in making a diagnosis of laryngeal paralysis.

Case No. 2. A clergyman, age 62, was admitted to St. Margaret's Hospital, December 7th, 1900. He was born in Germany, but had lived in this country many years. He had charge of a large parish in Kansas. His professional duties kept him quite busy. He was a strong man, presenting a good family history. He took cold easily, and had several attacks of influenza. They seemed to leave no bad results.

About a year before I saw him, he noticed that he was very short of breath and had some difficulty with his voice, which seemed to be very unreliable. When I saw him, he had just recovered from an attack of the grippe. He was weak and breathing with difficulty.

There was some impairment of his voice. The laryngeal examination revealed the left vocal cord in a partially paralyzed state. The thrill and pulsation were easily made out. The diagnosis of aortic aneurism was made. This patient was put on iodide and strychnine. This treatment gave him great relief. He was advised to lead a very quiet life.

Under the above treatment he became so much better that he decided to go South. I cautioned him, but he went to Asheville, N. C., where the altitude so aggravated his condition that he left and went to Florida. He remained in Florida for a few weeks only, most of the time he was in bed. He wrote to me that he would return to the hospital in Kansas City. He was taken so very ill at Birmingham, Ala., that he was removed to a hospital. The aneurism ruptured, and caused his death very suddenly while there.

These patients should be kept quiet on an easily digested non-stimulating diet. While their cases are always incurable, they can be made to live a very much more comfortable life. Absolute quiet should be insisted on. It is never safe to allow these patients to travel, because they are apt to die very suddenly, death being caused from rupture of the aneurism.

The laryngeal paralysis in both cases, aided me greatly in making a diagnosis.

This condition of the vocal cord was pointed out by Mr. Felix Semon, of London, years ago, in a very concise manner.

To-day it is known as the Semon law. Its importance should always be remembered in making a diagnosis in such cases as those cited above.

THE TONSIL SNARE.

BY W. H. PETERS, M.D., M.S., A.C., LAFAYETTE, IND.

Some years ago I presented to the profession a tonsil snare which had been in use for some years in my office. The snare had been thoroughly tested by several intelligent operators for a period of from eight to ten years, and the results had been so satisfactory that I considered them worth reporting. These snares have come into general use. Many thousands of them have been sold, but of late I have had so many inquiries in regard to the matter, and so many complaints, that I have thought best to give THE LARYNGOSCOPE readers the benefit of the answers which I have been compelled to write so often to individuals.

In the first place, there has been much dissatisfaction with the snares which have been made and furnished in the market, many of them having been made and furnished in the market, many of them having been made of cast iron, some of brass, and some of steel, so soft and inelastic that they either broke or quickly lost their shape. Most of those upon the market are altogether too large and clumsy, and not one of them in fifty, possesses the qualities which have made my original instrument so satisfactory in my own hands. The instrument is not a very large one. It should be made of the best of steel, very stiff and inelastic, and in every part should be free from any possibility of bending or yielding in use. To be satisfactory the snare must be absolutely rigid. Personally I have removed many thousand tonsils with this snare, and it has never failed me nor disappointed me.

The instrument may be described as follows: It consists of a handle, a canula with fenestrated tip, several of which are furnished with each instrument, and a screw adjustment, similar to that found on nearly all other snares. The screw adjustment in this instrument, however, has never been used by me, except for the purpose of taking up the slack wire before the operation. I use No. 7 piano steel wire, and here I will say that first-class wire is very difficult to obtain in the market. The best grade of wire is highly polished and very elastic, and possess very great tensile strength. There is no danger of it breaking. Before threading the snare, a loop should be formed upon the wire in the manner first suggested by Dr. A. T. Veeder of Pittsburg, Pa. One of the canulas should be inverted, and the two ends of the wire passed through it as

shown. A cork should be passed through the loop of the wire thus formed, the wires are then drawn in tightly, and shoulders are formed whereby the wire fits the fenestrum more perfectly. The complaints which have been made in the past, of inability to fasten the wire satisfactorily, have been entirely overcome in the present model. The ends of the wire must be very firmly twisted, not less than seven or eight twists being given them.

In an adult the operation is conducted as follows: Cocaine having been applied around the tonsil and in its crypts, five minutes are given for it to take effect. In the meantime the canula with the wire threaded has been sterilized, and the snare prepared for the operation as shown in the illustration, all of the slack wire

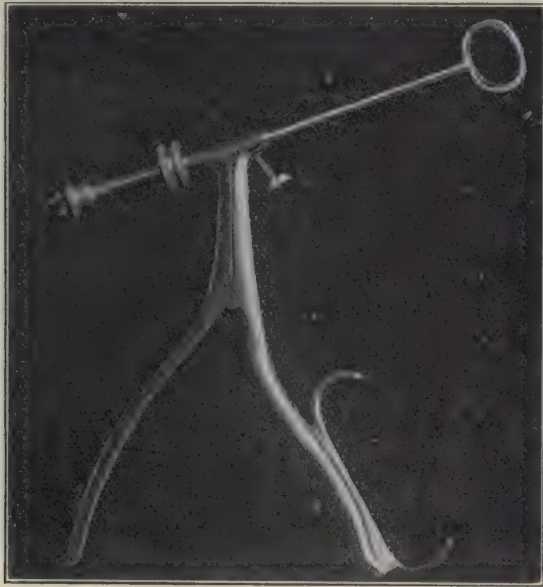


Fig. 1.

having been taken up by the screw adjustment. In an ordinary tonsil no forceps of any kind are necessary. The patient is seated in an ordinary operating chair, and the tongue is held down either by the operator or by the patient himself. The fenestrum with its guarded wire is pressed firmly down around the tonsil, when, with some manipulation, the tonsil will soon be seen to start forward through the loop. This movement of the tonsil, I have compared with the movement of the eyeball in the removal of an eye, when after the severing of the recti muscles, the speculum is pressed down into the socket, and the eye starts forward. At this point, the operator is instructed to sever the nerve with his scissors. Just so in the removal of a tonsil, when the tonsil starts forward into the loop,

the operator closes his hand upon the instrument, severing the tonsil with a single stroke.

The after treatment is very simple. The patient is instructed that crackers and milk, medium boiled eggs, and raw oysters can be swallowed with very little pain, and the patient is permitted, if he wishes to do so, to suck pieces of ice, holding them well back into the throat. The pain and soreness, however, are not very great, and I have never had a patient hesitate when it came to the removal of a second tonsil at a later sitting. As a rule, I remove only one tonsil at a time in an adult, and in the great majority of cases the tonsil is completely extirpated with a single application of the

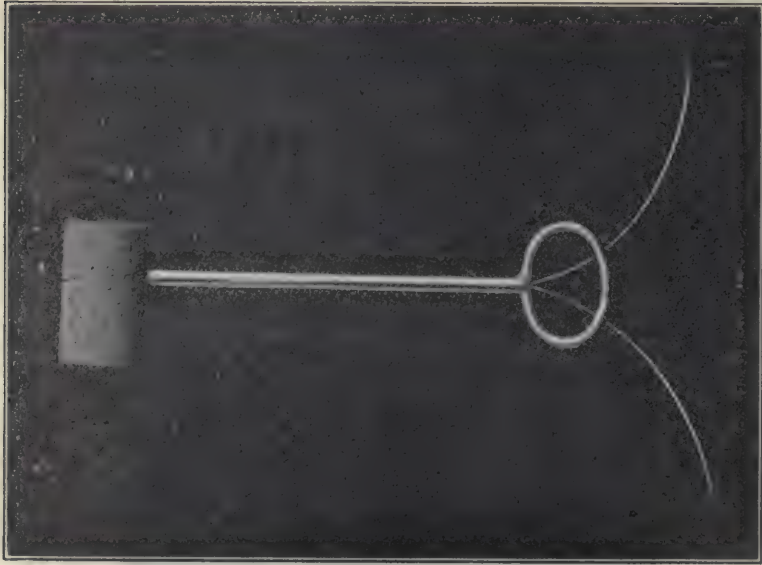


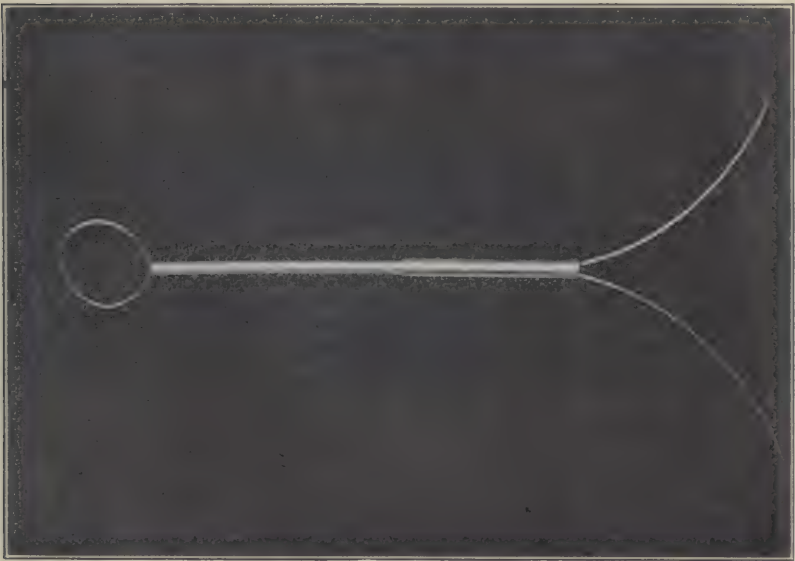
Fig. II. Showing the method of forming the wire loop.

snare. In some cases seizing forceps are necessary, and when so I recommend the forceps in the cut which were designed by myself for this purpose many years ago.

In little children I almost invariably use chloroform.. In these cases it is much better to have two snares sterilized and ready, and both tonsils are removed at the same time. I do not believe that the time which has been required in my office for the removal of both tonsils in little children under chloroform has averaged ten seconds. Immediately following the removal of the tonsils, with only such interval as is necessary to drop the snare and seize a suitable Pyncheon's curette, I remove the pharyngeal tonsil when it

is present. Every step of the three operations from beginning to end is a matter of but one or two minutes, and it is extremely rare for the child to be in my operating room more than fifteen or twenty minutes. The child is then ordinarily removed to a room adjoining, where, if so inclined, it is allowed to sleep off the effect of the chloroform, although in the great majority of cases the child is completely aroused within ten or fifteen minutes.

I have used these instruments constantly for over sixteen years. In my experience in children I have invariably found that under an anæsthetic the removal of both tonsils has left me a bloodless field, or practically so, for the removal of the pharyngeal tonsil,



• Fig. III. The straight canula.

while the removal of a single tonsil with the guillotine generally leaves the mouth so full of blood as to render the removal of the second tonsil somewhat unsatisfactory. In adults my experience has been similarly satisfactory. On one occasion a patient was brought to my office suffering from a secondary hemorrhage following a partially removed tonsil. The hemorrhage had intermittently lasted for two days, and the patient was in a serious condition. The removal of the remnant of the tonsil, made by the method described above, stopped the hemorrhage immediately. It has been said that the words "always" and "never" have no place in medical literature, and it is probable that there is no means by which a tonsil may be

removed that there is not some danger of hemorrhage. Dr. Balenger of Chicago has reported a serious hemorrhage in an adult from the use of this method and instrument. Such hemorrhages have, however, occurred from the slow ecrasement of the tonsil, and from the use of the galvano-cautery loop, and were numerous under the old guillotine and bistoury methods. Such danger following the use of the snare, however, is very remote, and may be practically disregarded.

Here I will make a suggestion. I do not know whether the procedure is new or not. I have used it repeatedly with the greatest satisfaction, when from any cause a hypertrophied tonsil has become inflamed, and its increased size renders it more troublesome. In syphilitic ulceration and in ordinary acute follicular inflammation of hypertrophied tonsils, I invariably remove the tonsil immediately, and have had as yet no reason to regret it. Also in cases of

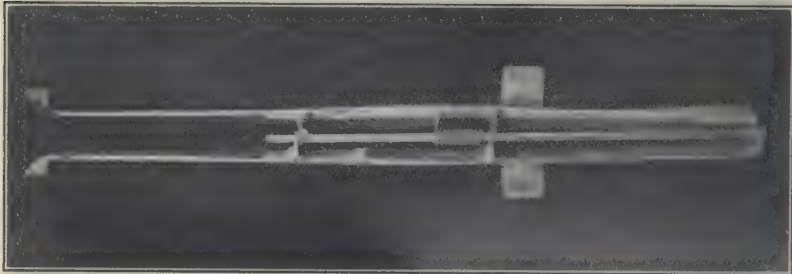


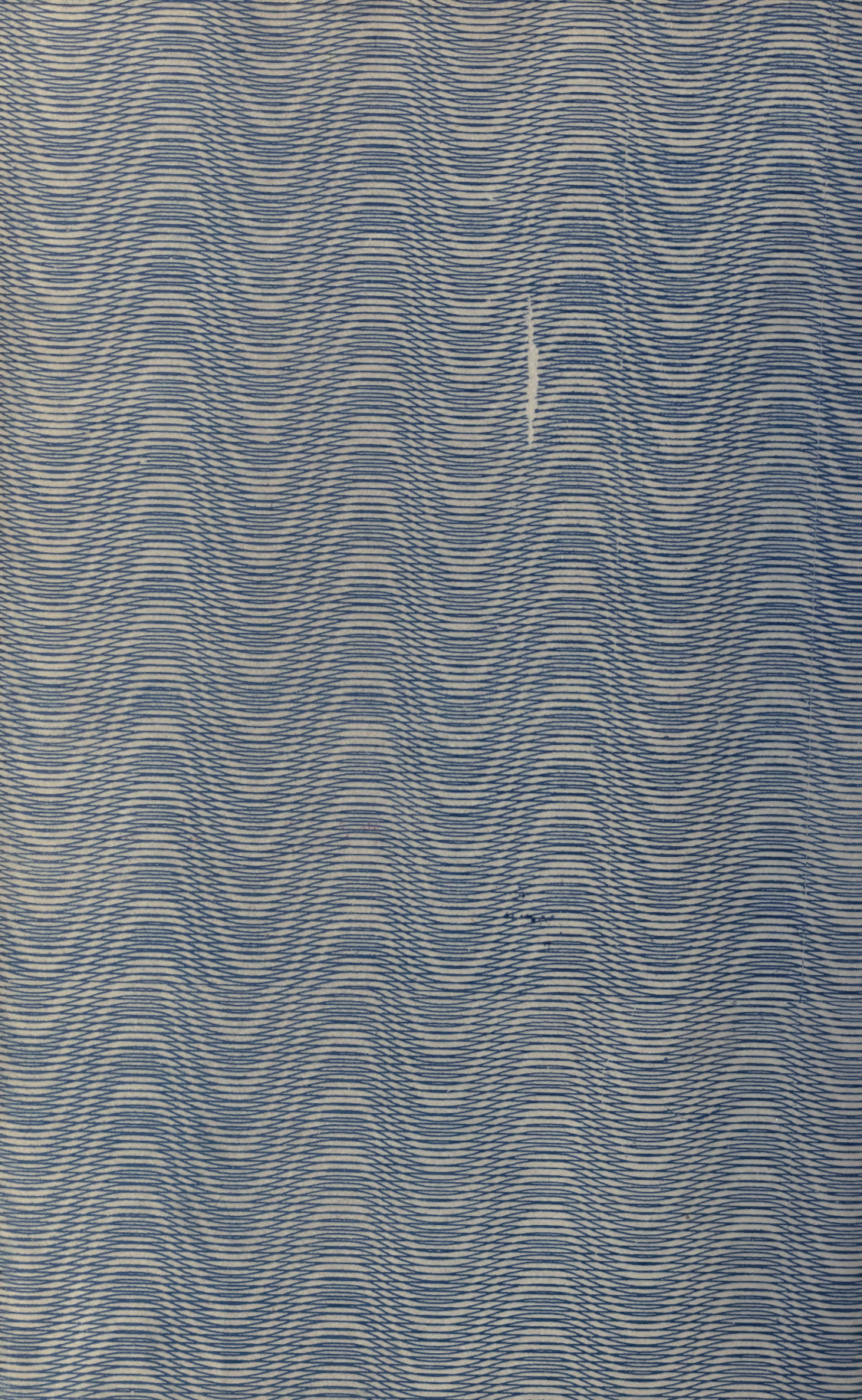
Fig. IV.

diphtheria, or where a child has been exposed to diphtheria, I have made it a practice to clear away the obstruction of the throat with very little ceremony.

In connection with this instrument, I wish to concede to Dr. Veeder the credit of using a fenestrated grooved tip for the tonsil snare. While my own work preceded his by some two years, still my snare was never satisfactory until his fenestrated canula came out. I have altered this canula at the present time, but he is still entitled to the credit for its originality.

In small and prominent tonsils, especially in adults, a straight metal tip in place of the fenestrated one has decided advantages. A wire should never be used a second time, and it is a positive advantage to keep the wires in a straight metal tube, rather than in coils.

I will be pleased to answer any questions in regard to these instruments or the method described. •



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